THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—34TH YEAR. SYDNEY, SATURDAY, FEBRUARY 1, 1947. No. 5.

Table of Contents.

CORONARY CLOTTING.

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RECENT experimental and therapeutic findings indicate that it may be necessary to modify the views commonly held on coronary thrombosis. These lines of investigation are three in number. In one instance perfusion of organs has given a lead with regard to the difference between organs in their ability to inhibit coagulation; in the second, treatment of patients suffering from coronary disease with a substance which lowers the coagulability of the blood has indicated the value of this treatment; and finally the most recent publication records the results of a pathological investigation indicating thrombosis as a factor in the pathogenesis of arterial changes.

Findings Accruing from the Perfusion of Isolated Organs with Diluted Heparinized Blood (Trethewie).

In work done on the perfusion of isolated organs, it was shown that when diluted heparinized blood was perfused through the isolated heart of the dog and the cat, the blood after passage through the heart was almost as coagulable as the blood passing to the organ, and only a negligible change occurred in the prothrombin and heparin content of the diluted plasma.[3] With other organs—for example the liver,[7] lung,[4][7] kidney[7] and spleen[9]—it was shown that the perfusion of the organ conferred anticoagulant effects upon the diluted plasma. In the case of the liver, fuller investigation showed that this effect could be attributed to an output of a substance in the protein fraction of the plasma. It was suggested that the freedom of such organs as the liver and kidney from thrombosis was due to this ability even with a slow rate of blood flow whereby anticoagulant effects were conferred upon the perfusing blood. It was further suggested that the lack of this property in the case of the heart might be responsible for the frequency of thrombosis in the heart, compared, for example, with the kidney, even though arterial changes might be great in either organ; and yet it is only the former which commonly shows thrombosis.

It was considered, in view of this work, that clot development might not be so much influenced by the arterial condition as by the coagulating effects. A paper then appeared from Baltimore which seemed to indicate that there was much to commend this view.

The Influence of Coumarin on Patients Suffering from Coronary Occlusion at Baltimore (Peters, Guyther and Brambel).

At Baltimore patients were divided into groups, one of which (60 patients) received the "accepted treatment" for coronary thrombosis. Fifty, in addition to this treatment, were given dicumarol, which reduced their plasma prothrombin level to 35% to 50%. The mortality rate—that is, the rate of patients who died in hospital—was in the control group 20% and in the treated group only 4%. Embolism was found in 16% of the untreated patients and in only 2% of the treated patients.[6] These figures are undoubtedly significant. The findings are so striking that they suggest that it is possible by varying hepatic activity (for it is to be remembered that coumarin injures the liver and reduces the output of prothrombin thereby) one can significantly affect the morbidity and development of embolism in cases of coronary thrombosis. This is a most remarkable piece of work and falls into line with the perfusion work, which indicated an inability on the part of the heart to inhibit coagulation to any extent, though a slight effect was observed.

Pathological Studies (Duguid).

Duguid studied cases of coronary thrombosis, and evidence was brought forward that many of the lesions classified as arterial thickening were in effect thrombi. It was further indicated that since red thrombi were prone

to softening and fatty degeneration, many of the appearances of atheroma might also be due to this process.[2]

Discussion.

It therefore appears that the condition of coronary occlusion may develop from changes not so much in the vessels themselves as in the coagulability of the blood or in the health of the heart tissue with regard to any slight anticoagulant activity it may have. It may be that changes in the vessels of a nature not allowing coagulation in protective organs (liver, kidney *et cetera*) may produce coagulation in the heart, since it is not a clot-protective organ. Certainly it is the case that once the condition is developed, the coagulability of the blood is important from the point of view of the outcome with regard to life and embolic phenomena. Now this is not so unreasonable a view as may be thought at first. This will be amplified later.

In a similar way experimental perfusion of the brain[8] has shown that not only does the brain not diminish coagulation of perfused fluid, but it may actually increase the coagulability of the blood. This is possibly the explanation why the brain also is so frequently involved in thrombosis while an organ with similar vascular disease such as the kidney is not. If this view were accepted, it would certainly be advisable to treat patients suffering from cerebral thrombosis with coumarin. So far there is no evidence in the literature that this has been done. The analogy is so obvious that this development also is obvious, and it would be better to use 3,3'ethylidene-bis-4-hydroxy-coumarin (E.D.C.), which is less toxic than coumarin.[4]

This observation falls into line with later work on increased coagulation of the blood produced by perfusion of the hind legs and pelvis,[10] which suggests a common basis for the occurrence of clotting in the hind limbs, heart and brain. An important parallelism is that thrombosis in the hind limbs begins in the veins of the muscles and not in the femoral or iliac veins.[10]

The importance of altering the coagulability of the in cases of femoral venous thrombosis has been well defined. Allen[1] has indicated that dicumarol in small doses is safe and effective as a preventive against thrombosis and embolism. It is useful for employment with femoral vein interruption after thrombosis has occurred. A convincing argument has been put forward by Bauer.[2] He states that thrombo-embolic complications occur in about 1·6% of surgical cases, and one-sixth of these patients die of pulmonary embolism. In association with pregnancy 1·2% of patients develop the condition and one twenty-fifth of these patients die. In medical wards there is a similar incidence of mortality. In lower limb fractures, the incidence of thrombosis is one-seventh. Bauer shows that heparin, an anticoagulant, is valuable immediately the diagnosis of femoral thrombosis is made; usually thrombosis begins in the deep veins of the lower part of the leg. Heparin is given in doses of 24,000 to 70,000 international units per day. In early cases this dosage usual aborts the disease. Recovery occurs in three to five days. In more pronounced thrombosis and pulmonary embolism, large doses prevent further clotting and usually carry the patient over the critical period, with rapid recovery. In Sweden there were only five deaths in 622 cases of thrombosis; thus the mortality rate among the patients treated with heparin was reduced to 1%, compared with 16% to 20% for surgical and medical patients and 3% to 5% for midwifery patients not receiving heparin treatment. The morbidity rate was also reduced to one fifth.

This brings us to consider that coronary thrombosis may be but a similar phenomenon to thrombosis of the vessels of the lower part of the leg. It is to be remembered that perfusion of the hind legs results in an increased coagulability of the blood after it has passed through the limb,[10] and this is assessed as prothrombin content, the increase being 24% in animal experiments. Evidence is available that the use of an anti-coagulant greatly reduces the mortality and morbidity rates in femoral thrombosis. Evidence is also available that similar treatment greatly reduces the mortality and morbidity rates in coronary thrombosis. As yet no one has given evidence that such treatment reduces the mortality rate in cerebral thrombosis.

Summary.

Evidence has been brought forward to indicate that thrombosis in the coronary arteries may depend more on factors influencing blood coagulation than on the condition of the vessels themselves.

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THE PITUITARY GLAND AND CARBOHYDRATE METABOLISM.

PART I. THE POSTERIOR LOBE.

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IN 1923 Burn[1] demonstrated that the subcutaneous administration of posterior pituitary lobe extract to a rabbit in hypoglycæmic convulsions would rapidly restore the blood sugar to its normal level. This observation has been applied clinically, and it is now a commonplace of therapeutics that a mild insulin hypoglycæmic state in man can be relieved by the subcutaneous administration of posterior pituitary lobe extract. Commercially several posterior pituitary lobe extracts are available; the term "Pituitrin" (Parke, Davis and Company) or "Infundin" (Burroughs Wellcome and Company) is usually applied to an extract containing both the oxytocic and pressor fractions. In 1928 Kamm, Aldrich *et alii* fractionated the posterior lobe extract into relatively pure vasopressor and oxytocic hormones, and these are issued by Parke, Davis and Company as "Pitressin" and "Pitocin" respectively. From the practical standpoint of therapeutics, there appears to be no great advantage in separating the two fractions. Posterior pituitary lobe extracts "obstetrical" and "surgical" are supplied by the Commonwealth Serum Laboratories. The former contains ten international oxytocic units per millilitre together with some of the pressor fraction, whilst the latter contains twenty pressor units per millilitre together with some oxytocic principle.

The observation of Burn on the effect of pituitrin on insulin hypoglycæmia resulted from a simple and clearly defined experiment, and one can safely state that mild hypoglycæmia in man or in the rabbit can generally be relieved by the subcutaneous injection of posterior pituitary lobe extract. However, the precise nature of the mechanism concerned is by no means clear and has been the subject of considerable controversy.

In view of the action of pituitary extract on insulin hypoglycæmia, one would expect to see some reflexion of this phenomenon in increases in the blood sugar content of man or laboratory animals after the injection of this hormone. It is doubtful, however, whether pituitary extract administered subcutaneously has any such effect; on the other hand there does seem definite evidence to indicate that the intravenous administration of posterior lobe extract leads to hyperglycæmia. In this connexion, however, there has been no unanimity of opinion whether the pressor or oxytocic principle is the responsible factor, and the relevant literature presents a mass of conflicting statements.

The position has been greatly clarified by the work of Holman and Ellsworth,[3] who have not only given an excellent summary of the literature, but have shown that in dogs the oxytocic principle is concerned with the production of hyperglycæmia. In their experiments these workers used highly purified and active pressor and oxytocic fractions termed postlobin "V" and postlobin "O" respectively. Postlobin "V" was 100 times as active as the standard pituitary powder and contained five oxytocic units per 100 pressor units, whilst postlobin "O" was 125 times as active as the standard powder and contained less than two pressor units per 100 oxytocic units. In dogs it was found that 0·0125 unit of postlobin "O" per kilogram of body weight would, if intravenously injected, produce significant hyperglycæmia. On the other hand, the injection of 0·25 unit of postlobin "V" per kilogram was required to produce a comparable effect. Holman and Ellsworth therefore concluded that the oxytocic principle was responsible for the hyperglycæmic action, and that any effect noted after the administration of the pressor principle was due to contamination by the oxytocic factor and in part to secondary circulatory disturbances.

Other investigators have studied the blood sugar level after the subcutaneous and simultaneous administration of pituitrin and insulin. Under these conditions it appeared that the hypoglycæmic action of insulin was inhibited. However, the true physiological significance of this phenomenon is somewhat uncertain, since there is the possibility that the pronounced vasoconstrictor action of pituitrin may delay the absorption of insulin.

In our own experiments we were more interested in ascertaining how the subcutaneous injection of posterior pituitary lobe extract exerted a beneficial effect on well-established insulin hypoglycæmia.

The early studies on this problem revealed some paradoxical findings. Burn[1] noted that the antagonistic effect of pituitary extract on insulin hypoglycæmia was "more persistent and much more rapid in onset than that of adrenaline". Further, pituitrin administered subcutaneously and simultaneously with adrenaline inhibited the hyperglycæmia which followed the injection of this latter hormone.

Lawrence and Hewlett[2] pointed out that the doses of pituitrin used in animal experiments were, in comparison with those given to man, grossly excessive. Thus in Burn's experiments the doses given to rabbits corresponded to 150 millilitres for an average human. Studies on man, normal and diabetic, following the subcutaneous administration of one millilitre of "Infundin" showed that this hormone exerted no appreciable effect on the fasting blood sugar level. Discussing the mode of action of the pituitary-insulin antagonism, Lawrence and Hewlett stated that since pituitrin inhibited the glycogenolysis due to adrenaline or anæsthetics, it was unlikely that it inhibited insulin action by stimulating glycogenolysis. Actually, however, ergotamine was found to abolish the pituitary-insulin antagonism, which would suggest that pituitrin counteracted insulin hypoglycæmia by mobilizing liver glycogen to glucose. It was therefore assumed that pituitrin antagonized insulin hyyoglycæmia by provoking the secretion of adrenaline, which in turn mobilized liver glycogen to glucose. These authors further suggested that the posterior lobe secretion played a complicated part in carbohydrate metabolism, in that it seemed to have a balancing effect in inhibiting the action of drugs which tended to alter the blood sugar concentration in either direction.

G. A. Clark[4] investigated the action of pituitrin in normal and spinal eviscerated cats with the suprarenal glands intact. In the latter the intravenous injection of pituitrin caused a fall in the blood sugar level. Clark attributed the hyperglycæmia that he observed in intact animals to a direct action of pituitrin on the hepatic cells, and concluded that the sympathetic system played no role whatsoever.

Lambie[5] carried out somewhat similar experiments to those of Clark, and observed that doses of pituitary extract that were sufficient to prevent insulin hypoglycæmia in the intact animal failed to do so when the liver was excluded from the circulation. In these experiments both pituitrin and insulin were injected subcutaneously, so that if we are to assume, as is suggested by some workers, that under such conditions the apparently antagonistic effect of pituitrin is due to delayed absorption of insulin, it is difficult to reconcile this theory with the fact that when the liver was out of the circulation the antagonism was no longer apparent.

Thaddea and Waly[6] observed hyperglycæmia in rabbits following the subcutaneous administration of ten units of tonephin.[1] This was not observed in animals with liver damage. Since a rise in the blood sugar level still occurred in adrenalectomized animals, these workers were of the opinion that the suprarenal glands were not implicated. Relevant to this latter statement is the work of Bischoff and Long,[7] who found that both pitressin and pituitrin administered subcutaneously to medullary adrenalectomized rabbits produced either hypoglycæmia or hyperglycæmia according to the state of the liver glycogen reserves. Further, when either pitressin or pituitrin was administered subcutaneously together with insulin to adrenalectomized animals, the hypoglycæmic action of the latter hormone was inhibited provided adequate liver glycogen was present.

Zunz and La Barre[8] have carried out detailed investigations of the action of the pressor and oxytocic principles on the secretion of adrenaline and insulin. Here again one is confronted with perplexing findings. The experimental procedure of these workers was precise; but one is puzzled to note that they found that pitressin injected intravenously into dogs under chloralose anæsthesia caused a rise in the blood sugar level. Under similar conditions the oxytocic fraction caused a fall, and in dogs with both suprarenal veins ligated the injection of either the pressor or oxytocic fractions resulted in a lowering of the blood sugar level. In some instances pituitrin was injected intravenously at varying intervals, and blood was taken from the suprarenal veins and tested on the rabbit's intestine for adrenaline content. It appeared that pitressin caused an increased secretion of adrenaline, whereas the oxytocic fraction had no such influence.

Perhaps this review of the work on the problem under discussion has been unduly prolonged; but in this work the reflective reader will appreciate the necessity for the meticulous appraisal of such experimental data. At least one fact appears to emerge—namely, that for the production of pituitrin hyperglycæmia, a liver with adequate glycogen stores is essential. It is not clear whether pituitrin acts directly on the liver or via the suprarenal glands, and in fact the only suggestive evidence that these may play a role is the work quoted above, that of La Barre.

Several workers have noted certain effects of pituitrin in rabbits. These we believe cannot be ignored; yet they are apparently not in accord with the results obtained in dogs. Ellsworth and co-workers[3] rather avoid the issue by stating that neither of the posterior pituitary hormones is of any significance in determining the blood sugar level in rabbits. From our own experience we consider that the rabbit comes into line with man, in that mild hypoglycæmia is rapidly relieved by the subcutaneous administration of pituitrin.

[1] A German preparation corresponding to vasopressin.

The following experiments were designed to determine the respective roles played by the liver and suprarenal glands in the pituitary-insulin antagonism.

Experimental Investigation.

For the most part adult cats under "Amytal" anæsthesia were used, although in some instances spinal or eviscerated preparations were used. "Amytal", as a 10% solution of the sodium salt, was injected intraperitoneally. The initial injection was 0·6 millilitre per kilogram of body weight, and when necessary this was supplemented by further doses of 0·2 millilitre per kilogram. Regular insulin (Commonwealth Serum Laboratories) was used, and the stated amounts were injected intravenously. The

TABLE I.
Experiment I. Normal Cat, "Amytal" Anæsthesia.

Time in Minutes.	Blood Glucose Content. (Milligrammes *per Centum*.)	
0	148	
30	145	← 5 units insulin
60	117	
90	92	
120	61	
a 150	55	
180	42	
210	34	
240	32	
270	34	
300	39	
330	34	
b 360	32	

dosages of insulin varied from three to ten units according to the initial blood glucose level and to whether the suprarenal glands were present. Blood samples, for glucose estimation by the method of Hagedorn and Jensen, were taken from the carotid artery. The posterior pituitary extract "surgical" (P.P.E.) was that supplied by the Commonwealth Serum Laboratories. All injections of this hormone were given subcutaneously.

Results.

The results were as shown in Table I.

This experiment demonstrates the degree and duration of hypoglycæmia that can be produced by a moderate dose of insulin. It will further be noted that during an experimental period of three and a half hours (*a* to *b*) there was no spontaneous rise in the blood sugar level (Table II).

These findings, together with those of the control experiment, indicate that the cat is a suitable animal for investigation of the pituitary-insulin antagonism. This

TABLE II.
Experiment II. Normal Cat, "Amytal" Anæsthesia.

Time in Minutes.	Blood Glucose Content. (Milligrammes *per Centum*.)	
0	120	← 5 units insulin
30	92	
60	57	
90	46	← 1 millilitre P.P.E.
120	59	
150	69	
180	81	

action of posterior pituitary extract "surgical" on insulin hypoglycæmia was confirmed on numerous occasions, but in some instances we were puzzled to note that the usual response did not occur. On investigation it was found that for posterior pituitary extract "surgical" to relieve insulin hypoglycæmia an adequate supply of liver glycogen was essential. This is illustrated in the following experiment (Table III).

After the taking of the last blood sample a piece of liver was removed, and on examination the glycogen content was found to be as low as 0·1%. This point concerning liver glycogen is confirmed in the following experiment (Table IV). The subject was an eviscerated cat under "Amytal" anæsthesia. The kidneys and suprarenal glands were intact. The rest of the viscera were removed, and although the liver was present it was not in the circulation. In order to maintain the blood sugar level, glucose was infused intravenously at the rate of sixty milligrammes per hour. When the blood sugar was at a steady level the experiment was commenced.

In the fifth experiment a normal cat was taken, and under ether anæsthesia the spinal cord in the cervical region was severed and the brain was destroyed. Artificial respiration was initiated, and after an appropriate interval had been allowed for the preparation to settle down, blood samples for glucose estimations were taken. When a steady state had been arrived at, the experimental procedures as shown in Table V were carried out. From

TABLE III.
Experiment III. Normal Cat, "Amytal". Anæsthesia.

Time in Minutes.	Blood Glucose Content. (Milligrammes *per Centum*.)	
0	142	← 5 units insulin
30	98	
60	62	
90	47	← 1 millilitre P.P.E.
120	39	
150	36	
180	40	

the above data it will be apparent that the higher nerve centres are not concerned in the pituitary-insulin antagonism.

Having established the fact that for posterior pituitary extract "surgical" to alleviate hyperglycæmia, an intact liver with adequate glycogen reserves is essential, we were next interested to know whether the suprarenal glands were essential for this reaction. To determine this, the antagonism between posterior pituitary extract "surgical" and insulin was studied in bilaterally adrenalectomized cats. This operation was carried out under "Amytal" anæsthesia. As a preliminary, the suprarenal veins were severed between ligatures and the glands were freed by blunt dissection from surrounding structures. By means of a series of ties encircling the glands it was possible to remove them in their entirety. After an appropriate

TABLE IV.

Time in Minutes.	Blood Glucose Content. (Milligrammes *per Centum*.)	
0	213	
30	215	← 10 units insulin
60	157	
90	106	
120	72	
150	54	
180	45	← 1 millilitre P.P.E.
210	43	
240	47	
270	43	
300	43	← 0·3 milligramme adrenaline
330	45	
360	47	

interval, at the end of which the blood sugar had attained a steady level, the experiment was commenced. The results are shown in Table VI.

This control experiment demonstrates that it is possible to produce in the adrenalectomized cat a state of hypoglycæmia which is suitable in degree and duration for the examination of the influence of posterior pituitary extract "surgical".

In this animal adequate supplies of liver glycogen were present, and further, as is shown by the injection of adrenaline, these were available to provide glucose for the blood; yet, in the absence of the suprarenal glands, posterior pituitary extract "surgical" was not able to relieve insulin hypoglycæmia.

The last of the experiments in this series was carried out on an adrenalectomized control; after adrenalectomy, the blood sugar level was observed for a period of several hours.

Discussion.

From the foregoing experiments it would appear that under the stated experimental conditions posterior pituitary extract "surgical" counteracts insulin hypoglycæmia by causing a secretion of adrenaline, which in turn accelerates the conversion of liver glycogen to blood glucose. Apparently posterior pituitary extract "surgical" does not, as some workers consider, act directly on the hepatic cells, nor is its effect mediated via the central nervous system. The latter was a possibility that had to be considered, since there is definite evidence that the hypothalamic region is involved in the regulation of carbohydrate metabolism. Lesions in the hypothalamic region may cause hyperglycæmia, and these may be interrelated with pituitary secretions.

TABLE V.

Time in Minutes.	Blood Glucose Content. (Milligrammes *per Centum.*)	
0	268	
30	274	← 10 units insulin
60	213	
90	145	
120	83	
150	52	← 1 millilitre P.P.E.
180	67	
210	79	
240	86	

It has been suggested that the secretion of the posterior lobe of the pituitary gland acts on the hypothalamic nuclei. Cushing[9] has shown that the ventricular cerebrospinal fluid contains a substance which behaves like pituitrin, and pituitrin when injected into the third ventricle acts immediately and more intensely than when administered subcutaneously or intravenously.

It is well established that the suprarenal glands afford a protective mechanism against hypoglycæmia. Their influence, exerted by the secretion of the medulla, mobilizes liver glycogen to blood glucose, whilst insulin

TABLE VI.

Experiment VI. Adrenalectomized Cat, "Amytal" Anæsthesia.

Time in Minutes.	Blood Glucose Content. (Milligrammes *per Centum.*)	
0	240	← 3 units insulin
30	219	
60	152	
90	110	
120	88	
150	72	
180	63	
210	59	
240	57	
270	54	
300	49	
330	39	
450	46	

decreases the blood sugar level so that the resultant effect will depend on the relative concentrations of the two hormones. In the experiments above described and in man with a fully developed hypoglycæmic state, it would appear that owing to excess insulin the normal balance is disturbed and adrenaline cannot compete with the insulin action. On the basis of this theory, however, it is difficult to interpret the action of posterior pituitary extract "surgical", and the only safe assumption is that it exerts an extra stimulus to the suprarenal glands and causes a further secretion of adrenaline.

The results of Experiment VIII would suggest that adrenaline is not the only hormone concerned in the maintenance of the normal blood sugar level. Thus, in the absence of the suprarenal glands, the blood glucose was maintained at a steady non-hypoglycæmic level for a period of several hours. One could assume that in such circumstances the cells of the paraganglionic system were able to take over; but it has not been established that these cells secrete adrenaline.

Gill and Lehmann observed that the addition of glucose to muscle extracts inhibited the enzyme which phosphorylates glycogen, and Soskin postulated the existence of a homeostatic mechanism in the liver for the regulation of the blood sugar level. It is assumed that the liver constantly supplies glucose to the blood, and that when the blood glucose level rises the liver compensates by stopping its supply to the blood. Conversely, when the blood glucose has again fallen to, or a little below, its previous normal level, the liver again resumes its output to the blood stream. Further, it is considered that whilst

TABLE VII.

Experiment VII. Adrenalectomized Cat, "Amytal" Anæsthesia; Liver Glycogen 5%.

Time in Minutes.	Blood Glucose Content. (Milligrammes *per Centum.*)	
0	141	
30	131	← 3 units insulin
60	92	
90	56	
120	47	← 1 millilitre P.P.E.
150	45	
180	45	
210	47	← 0·4 milligramme adrenaline subcutaneously
240	72	
270	89	

insulin is not essential for this homeostatic mechanism, the amount of insulin present and the amount of opposing hormones present determine the blood sugar level at which the regulating mechanism will work.

Against such a simple and attractive theory are the findings of Fantl and Rome,[10] who noted that only extremely high concentrations of glucose inhibited glycogen breakdown in liver pulp. These concentrations of glucose were outside the physiological range.

TABLE VIII.

Experiment VIII. Adrenalectomized Cat, "Amytal" Anæsthesia.

Time in Minutes.	Blood Glucose Content. (Milligrammes *per Centum.*)
0	113
30	106
60	110
90	115
120	119
150	117
180	119
210	124
240	117
270	119
300	111

Summary.

1. The pituitary-insulin antagonism has been studied in cats.

2. It appears that posterior pituitary extract "surgical" counteracts insulin hypoglycæmia by provoking a secretion of adrenaline from the suprarenal glands.

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NASAL OBSTRUCTION IN CHILDREN AND ITS RELATION TO DENTAL DEVELOPMENT AND GENERAL HEALTH.[1]

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IT is difficult to define the exact borderline between the scope of the ear, nose and throat surgeon and that of the dental surgeon in this discussion, so I have confined myself to enumeration of the common causes of nasal obstruction in children, referring to certain differences of opinion that exist.

I have indicated, shortly, the lines of treatment recommended for these conditions.

Causes of Nasal Obstruction.

1. *Congenital.*—(*a*) Choanal atresia, which may be unilateral or bilateral. It is usually membranous anteriorly, and bony and membranous posteriorly. (*b*) Septal deformities, which may not be truly congenital, but due to birth injuries.

2. *Traumatic.*—(*a*) Fractures of bone. (*b*) Fractures and dislocations of cartilage. (*c*) Foreign bodies: (i) rhinolith, (ii) anything that can be pushed into a nostril. (*d*) Septal hæmatoma. Since commencing this paper I encountered a case worth reporting here.

A girl, aged eight years, had been complaining of a bad smell and unilateral nasal discharge. The X-ray report was that of a dull right antrum and a hazy left antrum. Examination showed swollen right mucosa and a very indifferent airway on that side. Examination under an anæsthetic showed a swelling of the floor of the right nostril, and pressure on this produced thick pus. It was attached to the nasal floor, about half an inch inside the vestibule. On being grasped with Luc's forceps it came away without difficulty, leaving a small area of apparently intact bone on the nasal floor. Examination of the tissue removed showed an outer covering of mucous membrane. Inside this was a hard calcareous shell, and inside this again a tooth root with a granuloma on the tip.

To me, the obvious explanation is that the root became detached from the rest of the tooth and that the granuloma eroded through the bone and the pressure of new teeth forced the root through into the nose. I have not seen or heard of a similar case, and I would be interested to hear the comments of our dental colleagues. The right antrum wash-out contained a little thick mucus in the return, the left was clear.

3. *Infective.*—(*a*) Acute: (i) coryza, (ii) sinusitis, (iii) diphtheria, (iv) septal abscess. (*b*) Chronic: (i) congenital syphilis, (ii) sinusitis.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association and of the Australian Dental Association (New South Wales Branch) on September 26, 1946.

4. *New Growth.*—(*a*) benign—polypi. (*b*) Malignant—sarcoma, post-nasal particularly.

5. *Errors of Metabolism.*—(*a*) Allergy. (*b*) Hypertrophic rhinitis. (*c*) Adenoids.

It has been maintained that hypertrophied tonsils will cause nasal obstruction, but there is very little evidence to support this contention.

Treatment.

In congenital conditions treatment is surgical and immediate in all atresias. Septal deformities should be left until the nose has developed its adult shape and size, but if the obstruction is serious, then surgery must be employed to relieve it, even before full development is achieved.

Fractures and dislocations should be replaced as soon as swelling permits. Foreign bodies must be removed and septal hæmatomata opened and drained.

The details of treatment of the infective causes would take far too long to give in detail; suffice to say that the advent of chemotherapy has to a large extent altered the attitude towards the cases resulting from them.

All types of new growth should be removed surgically or by cauterization; in some cases, particularly when there is proof or a probability of malignancy, radium or deep X-ray therapy should be employed in an attempt to prevent recurrence.

We come now to the commonest and probably the most important causes of all; the group I have called "errors of metabolism".

Allergy, or paroxysmal rhinorrhœa, is a condition which is not very thoroughly understood, even now. There is no doubt that it is increasing in frequency, both in children and adults, and discussion of its multiple causes and largely unsatisfactory treatment would run into hours.

I shall confine myself to the following suggestions.

The modern tendency to create artificial atmospheres may have something to do with it; this is quite hypothetical.

There is a very strong nervous element concerned, which is more noticeable in adults, but there is no doubt that nervous, frightened children may get attacks of acute rhinorrhœa as soon as they visit a doctor or dentist. A lot of this can be put down to bad upbringing and the frightening of children by "bogeymen". Fortunately, this is a habit which is dying out, but there are still cases occurring.

There is another point I would bring to your notice. There is a quite definite association of apparently allergic conditions with sinusitis. Which is the cause and which the result, I am not prepared to say, but it is an association which is frequently overlooked, even by our own specialty.

Adenoids.

On looking through the literature it is a little surprising to find that there are marked differences of opinion as to the advisability of removing adenoids in children.

Taking two examples of articles published in 1932. Warwick James and Somerville Hasting[(1)] state definitely that adenoids do not cause nasal obstruction and that mouth breathing does not cause dental changes and deformities. They say that sinusitis is the only real cause of nasal obstruction in these cases, and that the fact that a child keeps its mouth open does not show that it is a true mouth breather. T. B. Layton supported them and said that it is very seldom that adenoids require removal. J. G. Turner[(2)] said that invariably he noted a widening of the dental arch following operation for adenoids, and he stated that, within reason, the earlier the operation was performed, the better the result.

These two views may be taken as extreme. The fact is, that every case must be considered on its merits and the vast preponderance of opinion is that adenoids should be removed when the situation demands it, and that they do cause nasal obstruction. I think it can be stated without fear of effective contradiction that the removal of tonsils and adenoids, in properly diagnosed cases, is one of the most beneficial operation known to surgery.

The chief difficulty is to determine if there is a co-existent sinusitis.

I will not go into the matter of differential diagnosis, but I do maintain that it is one of the most important possibilities and should always be in the mind of anyone who has to deal with children's health. If there is any doubt, it is quite easy to proof-puncture the antra while the patient is under the anæsthetic for removal of adenoids.

If a nose remains dirty after the adenoid operation, then there is a strong presumption of sinusitis.

In this connexion, for the benefit of the members of the dental profession, I shall try to enumerate the signs of nasal obstruction which can be seen without elaborate examination paraphernalia. There is no need to tell them about mouth breathing; they know as much as we do, and this will be dealt with thoroughly by Dr. Thornton Taylor.

Post-nasal discharge may be mucoid or muco-purulent, and can often be squeezed out by causing the patient to gag. Mucoid discharge is usually present with adenoids, and the muco-purulent type is more indicative of sinusitis. By tilting up the tip of the nose, the nasal vestibule can be examined and the following indications looked for: (*a*) Crusting and ulceration resulting from purulent discharge. (*b*) Dislocation of the anterior end of the septal cartilage. (*c*) Swelling or changes in colour of the inferior turbinates; in allergic conditions the mucosa tends to become pale and watery looking, or bluish.

The breathing capabilities can be tested by holding a small piece of cotton wool in front of each nostril in turn, while occluding the other and keeping the patient's mouth closed.

In conclusion I suggest that any mouth breathing may become a habit, even after the cause has been removed, and if this habit is not broken, then the nasal obstruction will continue and the nasal mucosa remain congested. For this reason it is important to relieve the obstruction as soon as possible and to institute nasal hygiene and breathing exercises until a satisfactory nasal airway is established.

This aspect is not appreciated nearly enough, even by members of our own specialty, and scarcely at all by the profession as a whole. In my opinion a proportion of the surgery done could be avoided if more stress was laid on the bad effects of mouth breathing, and parents were instructed better in the superintending of breathing exercises for their children.

Some of our hospitals have clinics connected with their physiotherapy departments for the teaching of correct breathing, but not all do, and even those which have them do not insist on all children with any form of nasal obstruction attending. To my mind this would be a very advisable procedure.

References.

(1) Warwick James and Somerville Hastings: "Discussion on Mouth Breathing and Nasal Obstruction", *Proceedings of the Royal Society of Medicine* (Section of Odontology), June, 1932, page 1343.

(2) J. G. Turner: "Some Clinical Observations in Connection with Causation of Deformities of Dental Arches", *Proceedings of the Royal Society of Medicine* (Section of Odontology), September, 1932, page 1643.

DENTO-FACIAL DEFORMITIES ASSOCIATED WITH NASAL OBSTRUCTION.[1]

BY A. THORNTON TAYLOR, D.D.Sc.,
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WE have listened with great interest and benefit to Dr. Bettington's dissertation on nasal obstruction from the rhinological viewpoint, and now we come to the consideration of another but no less important aspect of these disturbances and their sequelæ.

May I here pay tribute to the breadth of view of the chairmen of our respective syllabus committees for their understanding of the implications of our subject that they chose to associate an orthodontic concept with, may we concede, the more spectacular surgical specialty; for the orthodontist, while concerned with the ætiology of nasal obstruction, also plays a role in its treatment.

However, it is perhaps from physical considerations that this complementary paper can more properly be commenced, for commonly reference is made to the orthodontist when dento-facial deformity, to us a malocclusion, calls for functional or æsthetic improvement of those disharmonies associated with nasal obstruction.

Now how, in effect, are these disharmonies manifest? Do they follow any recognizable pattern or type?

While mouth breathing associated with post-nasal disturbance may occur with many differing types of malocclusion, two main types may be distinguished, differing broadly in ætiology and facial characteristics, with differing treatment problems and a varying prognosis.

Classification.

Broadly, one may classify a mechanical and a developmental type. It may be here suggested that hereditary factors may be involved in either.

Mechanical Type.

What I have termed the mechanical type is characterized by prominent upper incisor teeth, narrow dental arches, a backward or under-developed mandible with

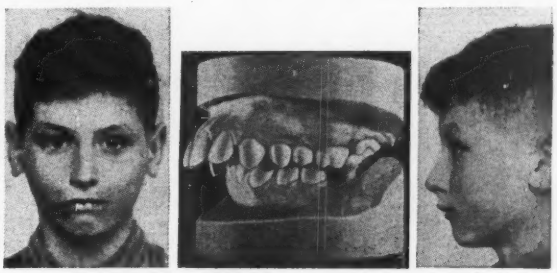


FIGURE I. (After Korkhaus.)

varying degrees of "bite-depth" or incisal overlap, depending very largely upon the ætiology of the condition. Broadly speaking, those types associated with mechanical causation, such as dummy or thumb sucking, tend to have open "bites", while the straightout mouth breather, by virtue of the backward position of the lower jaw, has a deep "bite", with the incisors commonly biting behind the necks of the upper incisors or further lingual on the palatal mucosa. (See Figure I.)

These malocclusions are progressive because from most simple beginnings they can develop to an ever-increasing degree of abnormality because of a vicious circle of muscular cause and effect acting to accentuate the disharmony.

Note that the upper lip is functionless and allows the upper incisors to move forward. The lower lip rests cushion-like between the lingual surfaces of the upper incisors and the labial surfaces of the lowers, thus continuing to press the uppers forward and hold the lowers back. The corners of the mouth press in, accentuating the narrowing of the dental arches, and in the early stages of the progression the lower incisors, by contact with the sloping upper incisors, tend to move the upper incisors further and further forward. The tongue is also lower than normal and does not perform its part in helping to maintain dental arch width.

These are the commonly accepted mouth breathing types and agree with typical textbook description.

Retardation Type.

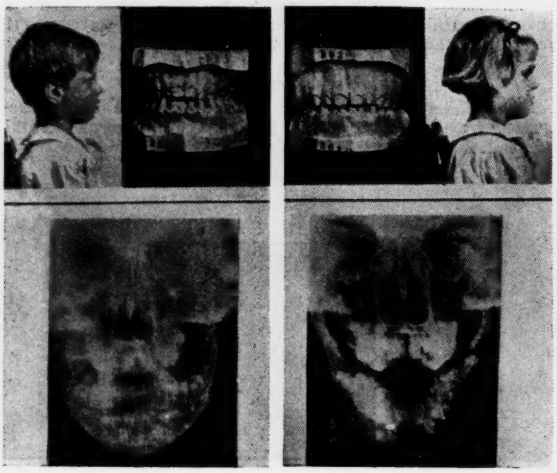
There is, however, another most distinctive type about which one can find little in current literature on growth and development. Rosenberger finds in his study of growth

[1] Read at a meeting of the New South Wales Branch of the British Medical Association and the Australian Dental Association (New South Wales Branch) on September 26, 1946. Dr. Taylor illustrated his paper by slides and models not included among the illustrations, comment on which is not published.

in the respiratory area that "disturbances of growth are divisible into two types. In one a normally grown nose fails to drift forward through failure of forward expansion of the cranium. In the other a forwardly drifting nasal skeleton itself shows disharmonic growth in its several dimensions."

In these types we have definite maldevelopment of the bony architecture of the naso-pharynx, either in whole or part, and the premaxillæ always, and the maxillæ in most cases, suffer in this growth lag, with consequent diminished dental arches, high palates, narrow nares, deflected nasal septa and other related defects.

The typical malocclusion that presents is usually marked by extreme irregularity; dental arch relationship may be normal, post-normal or mesial. The "bite", that is, incisal overlap, is usually shallow or open, although a deep incisal overlap may present because of the general lack of growth in the jaws. (See Figure II, (*a*). Contrast with Figure II, (*b*).)



(*a*) (*b*)

FIGURE II.

Contrast (*a*) with (*b*) (normal). Note deflection of septum in (*a*). (After Dewey.)

Some Factors of Significance.

Having considered very superficially the dental manifestations of mouth breathing, let us now pass on to discussion of some factors of significance.

Use and Disuse.

The most persistently perverse factor operating to continue mouth breathing, even when the airway is clear, is the functionless "hypotonic" upper lip—that spring roller blind that is ever up, as if it glories in displaying the pearls, not always dazzling, that lie beneath.

If you will examine the average mouth breather, you will notice that it is not the dropping of the lower jaw or the pendulous lower lip that holds the mouth open so much as the lack of tone and lack of development of the upper lip preventing it from coming down into apposition with the lower lip, which itself should close with the upper, meeting it above or gingival to the incisal edge of the upper incisor teeth. (See Figures III and IV.)

The biological principle of use and disuse is of the greatest significance in regard to the function of breathing. One may here quote Lamarch's third law as pertinent to our subject: "The development and efficiency of organs is constantly in proportion to the use of these organs." The functionless upper lip, similarly the loss of function of the nose as a respiratory organ, aptly proves the truth of Lamarch's dictum first enunciated in 1815.

For persistent mouth breathing children I like to tell the story of the well-worn bush track as an analogy to the use of their nose in breathing.

> As a little boy I had to go to school from my home through the bush and made use of a little bush track that led me to school. It was always nice and broad and I was able to hurry through it and get to school early. . . . At one time I got a little careless and went by another road that seemed easier and didn't use the usual track, except occasionally, and I found that it got choked with weeds and the bushes on each side grew closer together until I could not use the track at all. After a while someone told me that it was foolish of me to use the other road and that I should go back to the old pathway. So I tried very hard and boldly pushed my way through and went to school the old way. The next day and the next I battled through it and pushed all the weeds and straggling growth aside until soon the track was clear again. I was able to hurry along and found it so easy that I have never used the other road since.

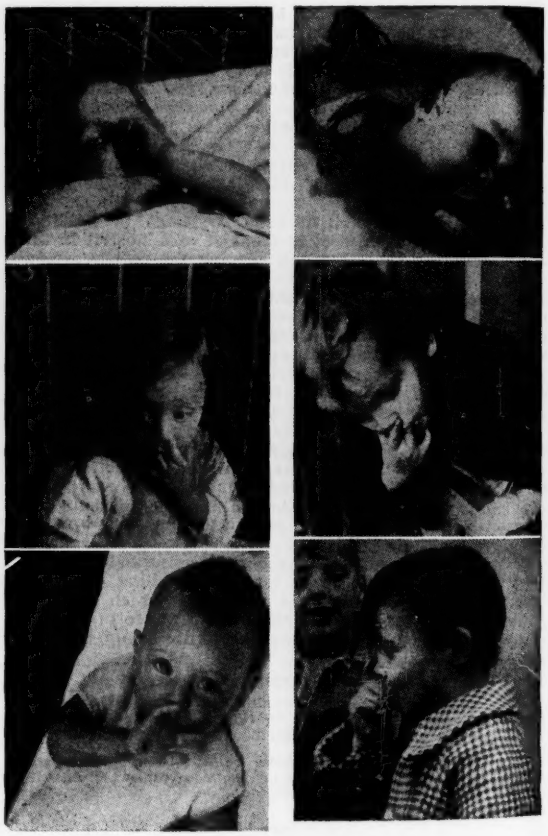


FIGURE III.

Some studies of finger-sucking habits in children, which are not necessarily harmful if not persisted in, but which, if associated with or followed by mouth-breathing tendencies, can lead to obvious dental arch deformity. (After Haas.)

One needs constantly to call on imagery and metaphor to carry our message to the children and their parents. Bald "do's" and "dont's" are of little practical help, for like so many other simple injunctions they are all too easily forgotten.

Heredity.

As the slides have perhaps indicated, and as your own personal knowledge and observation tell you, heredity can play a big part in determining whether or not abnormal respiration may be expected to occur or persist. Environmental factors also operate most strongly when there is an hereditary tendency to mouth breathing, because the child has no proper parental example, and also in many cases the parents have come to accept an aberration as normal. However, conscientious parents will endeavour to

overcome these factors and to do what they can to bring respiratory habits to normal. A defeatist attitude must here be countered on our own part especially, for it is no use saying that because a child has inherited an impaired growth pattern, we can do nothing for him. The factor that offers us most hope and satisfaction is the ability of bone to react to stimuli, both of use (by the patient) and mechanical by the orthodontist.

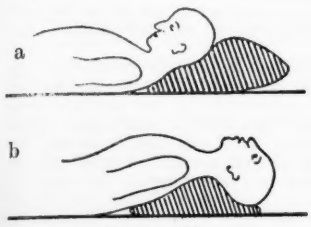


FIGURE IV.

Diagrammatic representation (*a*) of sleeping posture, exaggerated in (*b*), accentuating mouth-breathing tendency when the lower jaw is flexed open during sleep. (After Schwarz.)

Were not bone such a kindly tissue, our work would not be possible, not that we can grow bone—we do not claim that power—but we certainly can influence it to change. The teeth to us are merely handles by which we influence the bone, and dental arch expansion is possible only because the forces or stimuli, as we prefer to call them, that are applied through mechanical appliances attached to teeth, are transmitted directly to the bone beneath. It is only occasionally that one finds a bone that will not respond to stimulus.

With this heartening thought in mind, perhaps we may now turn to consideration of the means of treatment available to us.

Treatment.

The treatment of these conditions (whether or not they have passed through the rhinologist's hands) can be subdivided into a preventive and a facilitative phase.

Preventive Treatment.

Mechanical Treatment.—Preventive treatment, at our hands at any rate, embraces the use of mechanical appliances to render it difficult or impossible to breathe through the mouth. These, of course, can be used only when the airway is clear, or at any rate not completely blocked. They comprise essentially devices such as oral screens, the use of plasters to seal the mouth, bandages to tie it shut and the like.

Exercises.—Exercises are most difficult to describe, either in print or in such a static thing as a lecture, and a practical demonstration is needed to be of any value. Essentially, exercises may be grouped into: those that stimulate the forward movement of the jaw, that is, the thrusting type of exercise; the clenching exercise to bring the incisal planes of the teeth into function, so that the huge power of the masseto-temporal muscle group may be brought into play to lock a distal mandible into a forward position, providing dental arch harmony is attained in that position; the lip twisting exercise to develop power and tone in the *orbicularis oris* muscle group; lip stretching exercise to increase the length of the upper lip; breathing exercises *ad infinitum*.

For myself I pin my faith on what I term forced breathing, providing of course the nose can be used. In this I insist that the child perform some reasonably strenuous exercise compatible with his age, run or skip or hop or ride a bicycle vigorously until he is puffed, and do it for another five minutes with the mouth tightly closed. The increased respiratory pressure engendered by such action can be of enormous benefit if the exercise is done vigorously and conscientiously. (See Figure V.)

I have never yet seen contraindications to this practice, and I should like to hear in discussion whether I am endangering my patients in any way from such activities.

Facilitative Treatment.

Orthodontics provides a means to facilitate the closing of the mouth, and perhaps that constitutes the orthodontist's main sphere of usefulness in the treatment of the conditions under discussion.

It is of no use at all to expect a child to close his mouth and to keep it closed, if the physical contours of his denture are such that it is impossible for him so to do. Of course, as before mentioned, many patients in this category, through disuse atrophy of the muscles of their lips, are unable to keep the mouth reposefully closed, but these patients usually have dental arches in malrelation, and these are in fact invariably associated conditions.

(*b*) (*a*) (*b*)

FIGURE V.

Pictures of boy, (*a*) from class photograph in 1943, (*b*) in 1946, to show improvement in nasal development and facial muscle tone gained by conscientious performance of breathing exercises in conjunction with orthodontic treatment.

The orthodontist's province is to retract the prominent incisors or to expand both dental arches or both, and if necessary to bring the lower jaw forward as speedily as possible so that a degree of normal facial balance may be attained and the patient set on the path towards normal respiratory function. (See Figure VI.)

The question may arise in your mind as to the age when orthodontic treatment may be instituted and the simplest answer to this is to say "as early as its need is noticed". Most useful work can be done around the age of eight or nine in the mixed dentition, while the deciduous cuspids and molars are still present, when a relatively short treatment phase, never more than six months, is sufficient to facilitate normal breathing provided of course the patient cooperates in the elimination of the habits of malrespiration, previously described. Orthodontic treatment of established mouth breathers should never be deferred until the eruption of the permanent dentition. On orthodontic concepts, a second and final phase may be undertaken at the age of twelve, when the permanent dentition is complete, with the exception of the third molars. Those children—the majority—who gain materially from their earlier phase of treatment, need no more than a little adjustment to bring them to normal occlusion, while those who for multifarious

reasons either relapse or do not respond adequately, have at least learned the need for and value of cooperation and are better patients in the second stage.

The Psychology of Habit Prevention.

The elimination of perverse muscle habits and the building up of normal muscle function constitute without doubt the most serious problem that we are collectively called upon to meet, and this I venture to suggest constitutes more than anything else a psychological problem. Not mechanics, not surgery, but outright applied psychology offers the only means of successful resolution of the muscular disharmonies associated with mouth breathing.

I well remember one of the gravest problems in this field I was ever called upon to cope with—in relation to twins who were habitual mouth breathers. On my constant promptings to close their mouths they assured me they would look funny with their mouths closed; they were so conditioned to regarding the other twin (an identical twin I might mention) as normal that their mental conception of their own facial æsthetics accepted the open mouth as an integral and normal part of their make-up.

With all habitual mouth breathers there is undoubtedly a condition of muscular and mental unawareness of abnormality or aberration, because no great or obvious

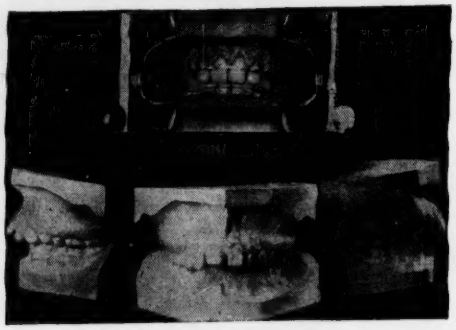


FIGURE VI.

Example of typical appliances used in treatment to retract prominent upper incisor teeth and open "bite" to facilitate closure of the mouth.

physical inconvenience is experienced, as is the case in muscle impairment associated with paralysis of the limbs from whatever cause, or in the graver types of poliomyelitis in which the respiratory muscles are unable to function. However, the need for muscle education or reeducation is just as real in mouth breathing as it is in these more inconvenient physical disabilities in which the nature of the disability is a constant and ever present reminder of grave physical infirmity to the sufferer.

In the naso-pharynx (denture) a vicious circle is all too commonly set up, but a definite and positive mental stimulus will create the tone and habit fixation necessary for permanent establishment of normal breathing habits, provided if in fact these habits are capable of establishment.

I feel fortified by a surge of enthusiasm to offer some criticism of rhinologists generally for lack of influence in regard to establishing normal breathing habits in patients who come under their care, particularly for surgical intervention.

While doubtless orthodontists can be accused of many sins of omission, I am obliged to say that my experience suggests that little or nothing is done by rhinologists and general practitioners working in this field to see that their patients develop normal breathing habits once their airway is made clear by surgical or other means.

This doubtless will be denied, and perhaps most hotly; but as the onlooker sees most of the game, I am reluctantly obliged to state that of hundreds of patients who come to us, either before or after rhinological intervention, few children or parents ever have an adequate understanding of the need for renewed and continued efforts on their part to establish normal breathing that has not previously been possible to them.

I have asked scores and scores of children if their "doctor" had told them anything about breathing or given them exercises and they invariably say no, and it is my contention here that if it is said, as I am sure it must be said, it is certainly said with insufficient emphasis and with no persistence.

Prognosis.

Prognosis of treatment depends largely on three factors: (*a*) the mechanics of respiration, (*b*) denture balance, (*c*) habit elimination.

Denture balance is of course intimately related to the mechanics of respiration; in point of fact they can scarcely be separated, but as our conception of the denture may vary, I have separated these two subject headings. Habit elimination is to my mind the dominant factor, for without it there can be no normal mechanics and without that there can be no balance in the denture.

ASTHMA AND OTHER PSYCHO-PHYSICAL INTERRELATIONS.

By CEDRIC SWANTON,
Sydney.

IN the past there has been a tendency to divorce the psychological from the physical side of medicine, and most of our efforts seem only to have widened this estrangement. The physical partner held himself aloof, proud of his physical accomplishments, unwilling to admit (perhaps even a little resentfully and scornfully) his need for and dependence on the emotional appeals of his rightful spouse. This psychological partner, withdrawn behind a barrier of phantasy, living rigidly within her narrow psychological limits, unhappily divorced, sensed the need for a closer union. Dynamic psychology pointed out the weaknesses in this separation and a partially satisfying union was achieved. This bore fruit in a none too robust but potentially healthy child which our American colleagues have rather unfeelingly christened psychosomatics.

It seems to me that there is no such thing as "functional" or psychological disease as opposed to physical disease. Perhaps it is a confusion of terms. Any functional or psychological response is inevitably physical, not only in its end result or symptom, but also in its production. To my mind, so-called psychosomatic responses depend on the fact that the autonomic nervous system, or rather, its cerebral "centres" (hypothalamus, basal ganglia *et cetera*), are linked up by pathways with the cortex and these centres thus come under cortical influence. This, of course, gives us some sort of imperfect control over our natural instinctive and emotional responses. These emotional responses with their physical concomitants are themselves physical and are supposed to arise in the base of the brain. The overt signs of these are expressed through the autonomic and voluntary nervous systems spontaneously, and are observed as specific physical reactions or as modes of behaviour. However, the cortical connexions of these ganglia allow some measure of voluntary (conscious) control over this autonomic behaviour (instinctive behaviour, if you like), but the control is very imperfect. As the cortical control is a late development phylogenetically, it follows that it is likely to be imperfect and unstable. Cortical control has developed because of our need to live as a partially civilized community, and so we must become socialized. This demands a training in our earliest formative years which is designed, necessarily, to give us some control over our more primitive demands. In the early years this is largely concerned with the need for control of our aggressive impulses. Aggressive impulses are produced as a direct result of the frustrations which are necessary to enable us to become socialized and to be able to live happily in a community rather than as omnipotent individuals. The adjustment to the control of these strong instinctive demands is brought about partly through fear and partly by our intellectual capacity to reason and

form judgements. This produces a constant battle for control between the id and the super ego, or if you like, between the autonomic centres and the cortex (depending on whether we are using psychological or physical language). It follows that this attempt at cortical control of our instinctive demands must be of a repressive or inhibitory nature.

Inhibition.

This inhibition is interesting. In the physical sense it represents what the psychiatrist refers to as suppression or repression. Suppression is conscious cortical inhibition and repression, which is unconscious, could be automatic or conditioned inhibition.

We now know that there are so-called suppressor bands in the cortex—areas that, if stimulated, produce inhibition. We could have deduced this from the fact that a reasoning person can have some control over his automatic or instinctive tendencies. For example, if we are frightened we do not necessarily bolt, nor do we always snarl or hit out if we are in a rage. This control is, as already stated, cortical, and must be activated by the mechanism of inhibition. We know of six suppressor bands or areas in the cortex which subserve an inhibitory function; there are probably more. For instance, there is area 2A which is just anterior to the precentral gyrus. If this area is stimulated a wave of inhibition spreads out and will alter or inhibit the response normally produced by stimulation of the precentral area. It is not difficult to imagine that the imperfect control of our emotional behaviour, referred to before, may result in disorders of inhibition producing an abnormal reaction of the autonomic nervous system. One can, amongst other things, conceive the idea of hysterical dissociations, fugue states, blindness, pareses, anæsthesiæ *et cetera* being abnormal inhibitory responses; and this links up the physical with the psychogenic mechanism.

It is suggested further that the mechanism of cerebral inhibition may be one of the most important factors in many so-called psychosomatic disturbances. We might expect to find types of personality with character development and behaviour responses peculiar to individuals suffering from certain disorders. I think that this is fairly plainly seen in the asthmatic, the stammerer, the ulcer group and others. The fact that cortical control of autonomic nervous system responses through inhibition does occur, is most easily seen in those systems which have been automatic functions at birth, but which have later come under some degree of voluntary control. These are the functions of respiration, defæcation, urination and so on. It is not surprising that under conditions of emotional stress, disturbances of these functions can occur. Perhaps the mechanism of cortical inhibition is most clearly demonstrated in the very common symptom of psychical impotence. Again, this inhibitory mechanism could also explain some of the anomalies of glandular secretions, of the abnormal motility of involuntary muscle, and various circulatory disturbances—for example, the normal sympathetic response of hypochlorhydria with lessened motility in the anxiety group, as opposed to the inhibition of the sympathetic with hyperchlorhydria and increased motility in the ulcer or "tension" group. To this tension group belong those people who are tense because of the necessity to exhibit the control which stimulates inhibitory responses.

Psychological Terminology.

We hear many scathing references to the jargon of the psychiatrist and the psychologist. I suppose this is inevitable as psychological theory has so far outrun anatomical and physiological knowledge that physiological and anatomical terminology does not exist to enable us to make satisfactory explanations in these terms. Unfortunately, this has helped to form a cleavage, as the physician is inclined to be impatient of the "jargon" and finds it difficult to accept something that is not expressed in the terminology that he understands. The psychiatrist, on the other hand, is often prone to forget that there must be underlying physiological mechanisms responsible for all his theoretical psychological reactions. (We have reached the stage latterly of teaching students of the mind-body interrelationships, which is an advance, but I do think that we should impress on the student that he is at all times dealing with an identity and not with a mind and a body. It is only in this way that all the factors in human abnormalities can be correlated to form a true clinical picture of these variations from the normal.) Dynamic psychology has given us satisfying explanations of our behaviour, and, what is more important, has enabled a form of treatment to be adopted which will relieve or cure a great deal of abnormal behaviour, which physical medicine cannot do. Were we less ignorant of the physiology of the brain, of its physical association paths and of all its many intricate functions, we could perhaps explain psychological mechanisms in physical terms. Perhaps it is a pity that Freud, carried along by a brilliant idea, did not remember his earlier physiological training and express himself in terms of cortical inhibition, autonomic response, thalamic rage responses and the conditioned reflex instead of repression, the unconscious, id reactions, identifications and so on. However, his terminology would have been so restricted that it would still have been necessary for him to coin a new "jargon" with which to explain his theories. The physiologists could not and still cannot give us sufficient evidence of paths and functions and physical mechanisms to describe so-called psychological behaviour in physical terms. As Freud said, in his Introductory Lectures: "The edifice of psycho analytic doctrine which we have erected is in reality but a superstructure which will have to be got on its organic foundation at some time or other; but this foundation is still unknown to us."

If only there were such things as unbiased people, then that which at present amounts to threats to one group and hopes of salvation to the other, would become nicely blended in an easy acceptance of both, and this might hurry us along the paths of advancing knowledge.

Having disposed of these generalizations, I shall try to show how personality difficulties with their abnormal responses can produce asthmatic attacks in subjects who possibly owe their disabilities to an inherited specific stigmatization of the autonomic nervous system.

The Asthmatic Diathesis.

It appears to be more or less generally conceded, and I think there can be little doubt of it, that those subjects who suffer from asthmatic attacks comprise a group who have a particular type of make-up, both physical and emotional, which predisposes them to these attacks. In other words, asthma is not a disease in itself, but is merely the symptomatic expression of a diathesis; or, if you like, there is a constitutional predisposition of the body as a whole to show certain local manifestations of this form of make-up in certain circumstances. What is this diathesis? Sir Arthur Hurst[1] sees no reason to change his original definition of asthma "as the reaction of an over excitable bronchial system, including the medullary centre, the vagal nerve endings and the bronchial musculature and mucosa to blood borne irritants and to reflex and psychic stimuli". I feel that this "buckshot" sort of definition is too narrow in that it confines itself to the reaction of the bronchial mechanism. I believe that the asthma diathesis consists of the tendency of a particular type of "identity" to react as a whole to its environment in a particular way so that under certain conditions of maladjustment asthmatic attacks are precipitated, and these are the evidence of an abnormal response of the individual. Or perhaps that these individuals have a stigma (probably inherited) of their central nervous system which predisposes them to exhibit a temperament with the development of peculiar personality responses; and that it is in the adaptation of these characteristics to their environment that they show the abnormal responses such as asthmatic attacks, allergic reactions and so on. In other words, these manifestations are end reactions of a central disability.

General Personality Characteristics.

The most striking thing about those who suffer from asthmatic attacks, and this is particularly noticeable in children, is that they all appear to have a particular type

of personality. Not only is this so, but the parent or parent substitute also shows very well-marked personality characteristics of a particular type. The characteristics in the personality of the asthmatic child have been variously described as nervous, irritable, excitable, aggressive, domineering, but also as over-anxious, frightened and lacking in confidence. Hurst,[1] for instance, quoting Rogerson,[2] says that the asthmatic child is over-anxious and lacking in confidence, whilst at the same time he often tends to be aggressive and dominating. These apparently conflicting characteristics readily fall into place if we look first of all at the personality of the preasthmatic child and then at the personality of the parent. We then realize that the full-blown asthmatic personality of the child is produced by a clash of these two distinctive personalities during the period of the child's growth and development. This is the stage at which the inhibitory factor comes into play.

The Preasthmatic Personality.

The preasthmatic personality of the child is undoubtedly nervous, excitable, irritable, aggressive and dominating, but changes are already noticeable before actual overt attacks are precipitated, because of the interplay of the child's personality with the domination and over-anxiety of the parent. And so it becomes difficult to tell just what characteristics belong to the child because of its make-up, or how far they are a reflection of the child's reactions to the parent. Psychometric investigations and play technique have disclosed that these children have "a high intelligence on verbal tests with a poorer performance ability, marked over-anxiety and lack of self-confidence, with considerable latent aggressiveness and ego-centricity". Rogerson,[4] basing his experience on the intelligence quotients of one hundred asthmatic children tested against a control group from the same class, assures us that, in general, asthmatics are above the average in intelligence, and further states that he has never seen asthma in a mental defective. I doubt if this means very much, as the figures are too small to have any real statistical value, but in my limited experience I have not seen an asthmatic child with a low intelligence quotient.

The Personality of the Parent.

The personality of the parent is invariably one of dominance over the child, but with a very well-marked, over-anxious and over-protective attitude as well. The parent is dominating, yet fearful for the child. The parent would no doubt be horrified at this description of herself, because she is really a very good person who is out to do her very best for the child; but she overdoes it. To the casual observer she does not appear to be a dominating type because of her fussiness and lack of confidence. However, she is unquestionably dominating, but in a very nice way! She is so afraid of any show of aggression in the child that she must try to stamp it out. She tends to be fussy, to have high standards, she is insistent on obedience, cannot tolerate disobedience, and is particular about tidiness, manners, honesty, truthfulness and so on. At the same time she is fearful for the child, afraid of danger for him, of illness, of his picking up bad habits, bad manners and so on. She has a rigid attitude towards bringing up the child. She has fixed ideas and standards of what she expects of children, but she is also anxious and always on the lookout for dangers. The same characteristics can frequently be found in her own parents. (Incidentally, this concern for the child is invaluable for therapy, as it means that we can usually get the full cooperation of the parent.)

Parent Child Personality Interactions.

It becomes obvious that the mercurial, irritable, excitable, aggressive and dominating preasthmatic child will clash with the dominating over-anxious parent. Most of the characteristics of the child irritate the parent who cannot tolerate domination and aggressiveness. Their personalities are too much alike. The parent, however, is bigger, wields the authority of the adult, is backed up by rigid ideas and standards of behaviour (gained, as a rule, from a similar upbringing), and the child is forced to go quietly and to become the good little boy. However, as we all know, frustration produces aggression and the child becomes resentful of this frustration. This resentment, if expressed, is considered by the parent to be bad and cannot be tolerated, and so more repressive measures are adopted. The child is unable to express himself freely, and not only has to hold back his aggressiveness, but he also becomes afraid of it. He becomes insecure, loses confidence in himself and is resentful of the parent's authority. He feels frustrated (suffocated, if you like) by the parent, who is quite unaware of the damage being done, as she is only doing what she feels to be right and in the best interests of the child. The child's resentment, only partially concealed in its behaviour, tends to increase the parent's over-anxiety and over-protectiveness. The child becomes fearful of his own aggressiveness, which he inhibits or represses, and he tends to become more and more dependent—but always against his will. His frustration creates an emotional reaction which his environment forces him to control, and I suggest that it is this inhibition of his natural emotional responses that is reflected in abnormal reactions of his autonomic nervous system which are expressed somatically as asthmatic attacks.

Personality of the Asthmatic Child.

So we see how the personality of the asthmatic child becomes a curious mixture of dependence with a demand for independence—how the child can appear to be aggressive and dominating and yet over-anxious and lacking in self-confidence, wanting to do things and to express itself and yet being too insecure to do so. He wants to be independent, but "he isn't let". The parent often says that the child is stubborn and obstinate, to which one can usually reply that perhaps it is a case of the pot calling the kettle black. These children frequently tend to be afraid of blood, accidents, hurt to others, have nightmares after seeing drama and reading gruesome books, and are upset by injustices. All of which, of course, represent fearful reactions to aggression. To my mind it is the inhibition, repression by fear, of his own aggressiveness during the so-called period of socialization that gives rise to the conditions which precipitate the asthmatic attacks. Further, any call on the child for a display of aggressiveness, to be independent and outgoing, can also be associated with anxiety in his mind and thus produce the inhibition which, acting through the autonomic nervous system, produces an "attack".

It is an interesting fact that there are many more child than adult asthmatics. The psychological explanation of this is, of course, that the child has become more mature and independent in his teens and tends to grow away from his parents and their rigid control, and is therefore less subject to parental frustrations.

Apropos of this, I once saw a boy who had suffered from moderately severe asthma until the age of fourteen. After explanation of his condition he was sent away to work for and to live with an easy going uncle. I saw him again ten years later when he told me that he had not had a recurrence of his asthma until three months previously. Six months prior to my seeing him he had married; and from the psychological point of view he had married his mother. His wife was over-anxious and over-protective, a fussy type who tended to nag, dominate and dragoon him.

Before leaving the personality characteristics of these children and their parents, I must admit that not all children with dominating parents suffer from asthma, nor do all vital, alive, aggressive or dependent children. It is only if they have these specific characteristics which I have tried to describe and which are part of the diathesis. There are, of course, degrees of apparent aggressiveness and dependence which vary with each particular child, but the personality picture is very clear. The distinctions may appear to be fine unless one has had a fairly close association and experience of these patients from the personality analysis point of view.

Physical Make-Up.

These personality traits are, to my mind, the most outstanding characteristics in the picture of the child asthmatic, but they do not, of course, tell the whole story. How much of this so-called preasthmatic personality is due

to inherited characteristics of temperament, and how much is due to specific physical defect and anomalies of autonomic nervous system responses I do not know; but the autonomic nervous systems of these patients do apparently respond to emotional disturbances in an abnormal fashion. For instance, it is generally conceded that in an asthmatic attack the "vagus system" from the medullary centre to the nerve endings in the bronchial musculature, or some part of this, is excited or stimulated or at any rate somehow gains ascendency over the sympathetic nervous system. Moreover, it is well known that injections of adrenaline, which we know mobilizes, activates, or stimulates the sympathetic to respond, will relieve an attack of asthma. It is interesting to speculate whether this so-called excitability or stimulation of the vagus is really responsible for the asthmatic attack, or whether it is not due to an inhibition of the sympathetic allowing overaction of the vagus. The ordinary emotional response to rage or excitement stimulates the sympathetic nervous system (Cannon[9]), but the emotional responses to aggression and resentment in the asthmatic—which are known to precipitate asthmatic attacks—apparently stimulate the vagus. This appears to be all wrong. However, perhaps the explanation of this reversal of autonomic response to emotional stimuli could be that there is an inhibition of the sympathetic nervous system, rather than a stimulation of the vagus. This could be cortical or at least cerebral. In other words, the necessity and demand for repression or inhibition of aggressiveness in the child actually produces cerebral inhibition of the sympathetic. Hence the over-action or action alone of the vagus precipitates the attack which one would expect could be relieved by reactivation of the sympathetic with adrenaline. And this we know to be so.

Here it might be interesting to quote the case of a boy, E.P., nineteen years of age, a student of singing. He had learned accidentally that his attacks subsided when he vividly day-dreamed that he was making his debut at the Metropolitan Opera House, New York, before a packed audience. This had the effect of normally stimulating his sympathetic nervous system which responded with an outpouring of adrenaline and with the subsidence of his attack.

Recent work on the inhibitory or suppressor bands in the cortex makes one wonder if cerebral inhibition of the sympathetic does not take place, and because of the distribution of these bands, if even selective cortical inhibition cannot occur. This is, of course, all highly speculative, but it could explain certain aspects of the problem of asthma. One also wonders, in view of the possible inheritance of the diathesis, if this inheritance is not what might be called a specific stigmatization of the autonomic nervous system, as would also appear to occur in stammerers. Perhaps there is also an analogy with that queer complaint which appears to be a disorder of inhibition—narcolepsy. May not this be a sort of inhibitory epilepsy, the somnolent turns being the sensory or *petit mal* attacks and the cataleptic episodes the motor or major inhibitory attacks?

Analogy with Stammering.

Perhaps the meaning of this mechanism of inhibition could be made clearer if we digress for a moment to the problem of stammering. There appears to be an analogy in asthmatics with a similar inhibition which seems to occur in stammerers. Stammering, to my way of thinking, has many similar characteristics to asthma, in that it appears to me to be an incoordination of the glottis associated with expiratory efforts which are due to psychological factors producing inhibition in those children who suffer from the stammering diathesis. For instance, a stammerer demands an auditor or he does not stammer. A child stammerer can talk to itself alone, read aloud alone, sing *et cetera* without a trace of a stammer. With an auditor he becomes inhibited. On the other hand, if he is spontaneously and normally aggressive, as in yelling for the ball in football, or shouting orders while gybing his spinnaker round a buoy in a sailing race, he does not stammer. In other words, his sympathetic nervous system is normally stimulated by a normal aggressive response, and there is no inhibition, cerebral or sympathetic. It is a different matter if, when he gets ashore, his commodore asks him how he got on in the race, or his headmaster asks for a ball-to-ball description of the match. Then his inhibition becomes all too obvious.

A case which may be of interest and which makes one think along these lines is the following.

A boy, E.C., of eighteen years, was one of four stammering brothers. Incidentally, they had a very strict, rigid, dominating father who ruled them with a rod of iron and of whom they were afraid. This boy, who had lost his own stammer twelve months previously, brought along one of his brothers and most aggressively demanded that I cure his brother of his stammer. When I asked the introducer how he lost his own stammer, he told me that a year ago he had suddenly got fed up with not being able to speak and decided that "what he had to say was b——— well worth saying and he was b——— well going to say it". And he had done so ever since without stammering. His brother was decently inhibited and retained his stammer!

Suggestibility.

One characteristic of the asthmatic is his suggestibility. It has long been recognized that suggestion can precipitate attacks and conversely that suggestion can be a potent force in relieving attacks. We all know that a person who is allergic to roses can be precipitated into an attack when he is confronted with a bowl of roses even though they be artificial; that an attack can be precipitated in a person who is sensitive to dust when he views a dust storm on the screen at the pictures; and that a patient can sometimes have an attack when he sees the motes floating in a sunbeam. I like the story of the French physician who used to have severe attacks of nocturnal asthma and who could get relief only by climbing out of bed, throwing up the window and inhaling the fresh air.

One night he went to bed in the usual fug that the French seem to like, with the window closed and the heater on. He woke in the middle of the night with a really severe attack of asthma. He felt so ill that he was unable to get out of bed to reach the window, so in desperation he leaned out of bed, seized his boot and hurled it, as he supposed, through the closed window. Gradually the severity of his attack lessened; it ultimately subsided and he went off to sleep. He awakened next morning to find the window intact and his boot reposing on the dressing table among the shattered remnants of the dressing table mirror.

Perhaps his aggressive act played an equal part with suggestion in relief of his symptoms. Suggestion in cases in which it precipitates attacks is probably a simple conditioned reflex response, in this case a conditioned response of inhibition. Curative suggestion, on the other hand, entails a belief in the suggestion, a blind acceptance of the practitioner's powers to cure. Confidence derived from the practitioner removes his inhibitions and bolsters up his own confidence, which is so important to the dependent asthmatic personality.

The Allergic Factor.

The allergic factor in asthma is of interest from the psychological point of view. Urbach, who, with Gottlieb,[6] has written largely on the subject of allergy, says that: "Everyone who has dealt with allergic patients is aware of the harmful influence of mental factors such as tension, conflict, fatigue, exhaustion, overwork, rush, disappointment, worry, stress and strain, apprehension, fear, grief, sex conflict, etc." He goes on to say: "In many cases of allergy it is impossible to effect a cure until the mental elements causing disturbances have been eliminated." It is well known, of course, that an offending allergen will cause allergic reactions on some occasions and not on others. Vaughan calls this "allergic equilibrium". Urbach writes that "there can be no doubt that allergic tolerance varies from time to time, depending on the conditions within the individual, depending also on contributory and precipitating factors . . . which may involve the endocrine glands, the autonomic nervous system, gastro intestinal resorption, infections, meteorologic or psychogenic influences".

Horneck found five times as many neurotic conditions among allergic as among non-allergic persons. Urbach remarks that "this would seem to justify the assumption

that nervous instability constitutes a predisposing factor in relation to allergic diseases". Perhaps we could say specific autonomic stigmatization.

It has been reported by Gillespie[6] and confirmed by others that in the treatment of allergic subjects by desensitization, the control groups "desensitized" by non-specific agents showed a similar and corresponding amelioration of symptoms. Clarkson[7] reported a case of an asthmatic girl who had a strongly positive skin reaction to egg. Under hypnosis the skin reaction was "negative". The next day it was strongly positive. There have been many similar results of the alteration of sensitivity by suggestion and these results have been confirmed. I remember an asthmatic girl who was afraid to go out of the house or even to have the window open for fear of wind-carried pollens, who was afraid to move for fear of stirring the dust of the house, and who was sensitive to so many foods that she lived on practically nothing but dry bread. This girl subsequently, without desensitization, ate what she liked and played golf on what is perhaps the windiest golf course in New South Wales.

It has been suggested (Urbach) (*a*) that the effects of the psychic factor produce an activation of the excitability of the autonomic nervous system, (*b*) that they bring about alterations in the local blood supply, and (*c*) that the effects are those of the conditioned reflex. It seems to me that all these three could come under the heading of abnormal reaction of autonomic nervous system control. It may be, for instance, that periodic resorption of unchanged protein by the gastric mucosa, producing allergic symptoms, could be a secondary result of abnormal changes of the mucosa, primarily produced through central abnormal autonomic nervous system responses. There has been an enormous amount of investigation of allergy and a great deal of preoccupation with the local manifestations, but I cannot help feeling that the allergic phenomena have a more general and perhaps central or cerebral origin and that the phenomena are merely the local effects of this.

The Infective Factor.

I feel that the infective factor in asthma is largely a secondary effect. The bronchial glands are stimulated to secrete by the overaction of the vagus or the inhibition of the sympathetic, whichever it is. Secondary emphysematous or bronchitic changes can and do occur. What part secondary infective factors play I do not know, although they can and do produce a lowering of the general resistance of the patient.

There is always a lot of talk about nasal and sinus infection. R. L. Cecil[8] says that: "About half our cases are found to have lesions of the paranasal sinuses varying from thickened membrane to a full blown pan-sinusitis of the polypoid type and most of them have already had operations on noses or sinuses, obviously without relief. The high proportions are good evidence that these lesions are part of the picture of asthma and not the cause of it." Asthmatic children are frequently nosey children. They have pale, swollen, wet-looking nasal mucosa and a condition that I think can only be referred to as allergic rhinitis. The precursor of an attack of asthma in these children is often said to be a cold. This is really an abnormal response of the nasal mucosa due to autonomic instability, or, if you prefer it, an attack of allergic rhinitis. Owing to this unhealthy or abnormal condition of the nasal mucosa, sinus infection could occur secondarily and, no doubt, frequently does. I think that it is rarely primary, although there is no reason why an asthmatic should not have a sinusitis like anyone else.

I have been informed that the ear, nose and throat surgeon shudders when he sees one of these children. He recognizes him when he walks in his door and tends to refer to him as the "only child" type, the type who is brought by both parents, the spoilt child or the over-protected child with the fussy parent, the wet nose type and so on. An ear, nose and throat surgeon told me recently of one of these children on whom he had scrupulously refused to operate, but finally on one occasion, because the child was apparently so ill and had a rise in temperature, he was forced to wash out his antra against his better judgement. The child recovered from this episode, but remained the victim of allergic rhinitis for a year or two. Subsequently the surgeon met the child's mother. She was no longer anxious, her face was wreathed in smiles, and when asked about the child she said that he was perfectly cured and had lost all his nasal and chest symptoms. On inquiring how this miracle had been accomplished, the surgeon was informed that it was by the simple means of sending the child to boarding school in the country. The change of air, you know!

It would seem now that antrostomy is becoming out of date for children of this type, and, as one ear, nose and throat surgeon remarked, perhaps operations of this nature for conditions of this sort have been responsible for the opinion so frequently heard from the lay public these days to the effect that "no one is going to touch my nose"! The more modern treatment is with drops and ephedrobarbital, a reasonable treatment, as it is presumably an attempt to hit the cause and at the same time shrink the local condition! I doubt if anyone can have very much to do with children without becoming aware of the existence of the nasal, frustrated, over-protected child and his proneness to asthma and hay fever.

Meteorology and Geography.

In regard to climate and locality, Price[9] says: "Asthmatics seem very sensitive to both of these, but no general relationship can be proved as the effects are most variable. Some patients are better in dry, others in damp, foggy climates, and in regard to locality each patient is a law to himself."

I have been impressed by the cases of two children who were each relieved of their asthmatic attacks, one by being sent from a seaside suburb to a country town to live, and the other one being sent from the same country town to a city suburb. Each child had dramatic relief from attacks, and naturally it was accepted that the seaside air was no good to one child and that the country atmosphere was no good to the other. Actually the atmosphere was entirely changed for both children, but the difference was not meterological or geographical, but psychological. In one instance the child was sent up to a placid and kindly grandmother in the country, and in the other the child was sent down to an easy-going and affectionate aunt. A visit by the parent to the child in the country was associated with a recurrence of symptoms and attacks.

In writing of spa treatment, Sir Arthur Hurst[10] (a self-confessed asthmatic) writes: "My own experience at these resorts convinced me that they acted entirely by suggestion. At Mont Dore one drank the water, inhaled its vapour, and to make doubly sure had a daily footbath in it. My first visit did me good. On my second visit I went without my wife and I hated it. It made me worse, but I got quickly well when I rejoined her in Paris. The psychological atmosphere at Bad Reichenall was so unpleasant that I did not cease being asthmatic till I left for Pontricina, where I was at once able to walk and climb all day." Bad habits die hard! Anyone who knows anything of psychology realizes that a person suffering from a psychological disorder has no insight into the underlying mechanism of his symptom. For example, the claustrophobe knows that he gets panicky under certain conditions; he knows that it is silly perhaps, that it is a psychological anomaly, but is quite ignorant of the reasons underlying the symbolism of his symptom. Perhaps it is not too far fetched to suggest that there is very often far more than suggestion underlying this relief of attacks by visiting foreign spas, removing children to the country and so on. Incidentally, in the psychological examinations of a collection of asthmatics, suggested by Professor C. G. Lambie but halted by the war, fourteen of sixteen asthmatics examined admitted that they suffered from claustrophobia (fear of being shut in, symbolic of being dominated, frustrated, held down, with the associated repression or inhibition of aggression).

Treatments.

Supposedly specific treatments for asthma have been legion. These different treatments have had their advocates and have undoubtedly produced relief, temporarily anyway,

and each in about the same percentage of cases. The fact that not one of these treatments has been generally adopted indicates, of course, that none of them are specific. Because of the widely different approaches of the various therapists and the fact that some appear to have no logical basis for their rationale, it appears that the personality of the physician, the patient's confidence in him, and suggestion (all of which produce a changed attitude of the person as a whole) are the common factors in many cures. In other words, they spell security and protection for the patient. As a colleague once remarksd: "If you've got enough fireworks you can cure asthma with anything from blue lights to fish hook enemata!"

Desensitization.

It has been shown by many and confirmed by others that improvements from symptoms occur with about equal frequency whether specific or non-specific material is used to desensitize the patient. Actually, to be most effective, I feel that this treatment should always be done by the enthusiastic physician himself, and not relegated to the nurse to do in a humdrum routine fashion. The results appear to be directly proportional to the amount of enthusiasm and confidence that is injected with the specific material.

Change of Locale.

I think that the change of locale is covered by my previous remarks and that change of the psychological rather than the meteorological or geographical atmosphere is the more important of the two.

Shock Treatment.

Insulin shock treatment has recently been recommended by Z. Godlowski.[10] The results in a very small series of cases have been published. These are too few to have any real value. However, the author suggests that insulin shock "operated by stimulating the adrenal medullæ inducing hyperproduction and dissipation of adrenaline: this action must be regarded as a defensive measure of the organism itself against the toxic action of massive doses of insulin". At least this form of therapy has the merit of having some rationale based on its supposed adrenergic effect. That it can produce a permanent change in the response of the autonomic nervous system seems unlikely.

The Whiteman Treatment.

One of the most recent treatments being investigated at the present time in a special asthma clinic at Royal Prince Alfred Hospital postulates that the cause of asthma is nasal infection. As a preliminary to prolonged nasal toilet, double antrostomy is performed in all cases. The patient signs and receives a copy of these instructions:

> I have read and am to receive a copy of instructions for patients treated at the Royal Prince Alfred Hospital Special Asthma Clinic, and quite understand:
>
> 1. That it is not suggested that this is a "cure" for Asthma, but it is an effort to show the Cause of Asthma, and how Patients can get and keep themselves better.
> 2. That the treatment is for Nasal Infection and no result can be expected under three months, the greater part of which will be spent in bed, and no security from attacks under twelve months at least, and then only if the one rule of treatment is carefully carried out.
> 3. That the one and only rule of treatment is:
>
> At any sign of the return of your former symptoms, or at any sign of a cold, or any indication of any trouble, or of more discharge in your nose or throat, you will immediately go to bed and inhale, and ring up Sister-in-Charge of the Special Asthma Clinic at the Hospital (Telephone LA 0444—Extension 241), and stay there well covered up until she decides you are well enough to get up.
>
> 4. I quite understand that failure to carry out the above rule exactly is failure to carry out the treatment.
> 5. And that, even when better, colds have still to be completely cleared up, and if allowed to run on, catarrh will become reestablished and symptoms return.

It would be interesting to compare the results of cases treated by antrostomy with the usual careful after toilet and cases treated by the "Instructions" alone. I think we know that antrostomy alone does not relieve patients of asthmatic attacks even with the most perfect technique and after toilet. Further, we know of cases in which nasal infection cannot be demonstrated. Consequently it seems a pity to inflict antrostomy on these patients. We know also that certain non-operative regimes conducted confidently and with assurance will also relieve attacks. However, the interesting thing is that some of these patients are relieved of their attacks if they follow the instructions, and a preliminary report on these cases states that "the majority of patients derived substantial benefit from the treatment. . . . The results compare very favourably with other recognised methods of treatment in showing the patient a new way of life". With a psychological understanding of asthma one might even expect a higher percentage of patients to be relieved, as I cannot help feeling that this treatment achieves its results entirely psychologically. However, to my mind it is very bad psychology indeed, as it places all the emphasis on the dependent attitude of these patients' personalities and on the need for dependence in their conflict. If they have the smallest difficulty, anxiety or frustration producing the slightest so-called nasal allergic reactions, or at the first onset of wheeze, they are instructed to go to the sanctuary of their bed, immediately to send for the nurse (mother), and to stay in bed until she says they can get up. I hope that for economic reasons alone this "new way of life" will not be extended to the rest of our anxious community! Many patients, the dependent ones, will submit to this form of therapy. But we have seen that these people are for the most part a curious mixture of dependence with a demand for independence, and in the less repressed this independence runs counter to the frustration implied in the treatment. I think that this is bad psychology because it is pandering to the defensive mechanism, or the running away side of the asthmatic personality, and only makes him more dependent on the doctor, the clinic and the nurse and tends to turn him into an economic misfit. In other words, it is a regression. How much better to be constructive, and to foster the outgoing aggressive independent demand in the asthmatic personality with encouragement to stand on his own two feet, and to become a mature adequate person in full control of his emotions and free from the anxiety that helps to precipitate his attacks.

Treatment of the Asthmatic Attack.

The treatment of the actual asthmatic attack is generally well known and consists of an attempt to relieve the bronchial muscle spasm. This is best achieved by stimulation of the sympathetic nervous system with adrenaline. Three to five minims of a one in 1,000 solution will relieve mild attacks, and the continuous adrenaline technique can be used in more severe cases. Aminophylline has been used recently and is usually given *per rectum*. Various other drugs have been used, and a useful summary of present day treatment of asthmatic attacks was published recently in THE MEDICAL JOURNAL OF AUSTRALIA.[11]

General.

Whatever treatment is instituted for an asthmatic, I feel that it must be inadequate unless the patient is viewed as an identity or person, an identity who is reacting as a whole to his environment in an abnormal fashion. Even if we accept the fact that he has an inherited handicap of diathesis or autonomic stigmatization, we do know that he reacts abnormally with his personality to certain external and internal demands. We should therefore in all cases attempt to alter his attitude to himself, and secondly, attempt to alter the external factors which, in the case of children at any rate, produce the inhibitions which retard his normal growth and maturation and prevent him from developing into an independent and adequate person.

Psychological Treatment.

In the case of children psychological treatment is almost solely a question of parent education, and the treatment should fall within the sphere of the family psysician. As in most of the nervous disorders of childhood, it is rarely necessary to see the child. In fact, it is quite dramatic to see a child's asthma "cured" by one or two sessions with a cooperative parent. Incidentally, this surely disproves the contention that the "cure" comes under the heading of suggestion.

If we do see the child, we do no more than tell him quite confidently how glad we are that he is now growing up and getting stronger, and that we are quite sure his troubles are over and that his attacks of asthma will soon cease. We convey to him the impression that the attacks are quite unimportant and that we are completely unconcerned about them. In fact, it is far better not to discuss them with the child at all, but just to have a talk to him on general lines about his interests, friends, games school *et cetera*, and then casually, as he is leaving, drop the remark that this chest of his that his mother is so worried about is perfectly all right, that he is growing out of his attacks, that they are unimportant anyway and will soon cease.

In dealing with the parent, we are on easy ground if we recognize the personalities of parents and children as described in the earlier part of this paper. These are so apparent as to be unmistakable.

Our aims are firstly to break down the over-anxious and over-protective attitude of the parent to the child, and secondly to make the parents realize just how dominating and demanding they are, and thirdly to point out to them the inevitable behaviour responses of this type of child to this sort of handling. We can start off by giving the parent a very simple little explanation of the part that emotional factors play in symptom formation in general. It is very important not to blame the parents, and to make them feel more anxious and guilty about their previous attitude to the child. They are already anxious enough, perhaps fortunately, as this makes them likely to be cooperative. We must not knock all their props away at once, but leave a few strategically placed before we knock some of them over.

For instance, knowing that the child is an asthmatic, we can gain the parents' confidences by describing quite confidently the child's and the parents' personalities. We then go on to tell them that these attacks occur particularly in intelligent children and that these children tend to have more initiative, drive and resource than most—that we realize the parents of asthmatic children are good parents, that they have very high standards and that they are out to do the very best for the child and so on. We then suggest that perhaps the parent is a bit more dominating than he suspects; that perhaps the standards he is demanding of the child are adult standards and that he is expecting a bit too much too soon; that we cannot expect politeness and good mannners, implict obedience and so on all at once; that the child will achieve these in time through our example rather than by being nagged into it; that lecturing and dragooning never work; that you cannot reason children out of emotional difficulties. We try to emphasize the fact that we should be more inclined to let children grow up in easy stages rather than to bring them up. We can explain at this stage that frustration produces aggressiveness, and that although the child may be inclined to be obstinate and stubborn, this is not innate in the child but a reaction of the child to the parent's rigidity, obstinacy and stubbornness. We point out that outgoing tendencies, egocentricity, resourcefulness and even aggressiveness are useful characteristics in building a good personality if one is not afraid of them and if they are usefully directed. Further, we point out the danger of over-anxiety and over-protection. We can show the parent that these children are spoiled children, but in a different sense to the spoiling that the parents understand. For instance, we say that one can spoil children only by domination and by expecting too much too soon, or on the other hand by being over-anxious and over-protective and not allowing the child to do what he is able to do when he is old enough to do it. Children can become independent only if they have a background of security, and over-anxiety and over-protection only convey our own anxiety to the child, and make him anxious and fearful with a tendency to clinging dependence. If we smack him down and do not let him do the things he is old enough to do when he is old enough to do them, then he will always be dependent and inclined to lean on us. We point out that asthma is the physical expression of his emotional tension, and expresses his frustration or anxiety in being independent. We can even call it parental suffocation. We can go on to point out that the child's asthma tends to make the parents more anxious, and that this leads to an increase in solicitude and makes them even more careful and meticulous for his welfare. The child tends to realize (subconsciously—and often not so very sub) that the only time the parents are really on his side is when he has asthma. He sees that their dominating attitude disappears like magic when he becomes sick, and that his symptom hits the parents where it hurts most (as indeed do most children's nervous symptoms). A neurotic symptom is a compromise. Teleologically the asthma satisfies both sides of the conflict in that it expresses his aggressive domination of the parents but at the same time makes him more dependent and wins their sympathy. Finally we tell the parent, or parents, that there is really no need for the child to have these attacks if they can recognize how their own personalities clash with the child's, and if they are prepared to do something about it. We impress on them the importance of adopting an impersonal attitude towards the attacks, and insist that these children do not have to wear scarves, mufflers and three singlets and be prevented from playing football.

Above all, we try to impress on them that it is far better to educate the child to accept responsibility rather than to protect him from it, and that their best efforts so far have only served unwittingly to hold him back.

Cases.

These are two cases selected at random; one patient is a child and the other an adult.

Case I.

Betty T., aged ten years, had suffered from asthma from the age of three years. She had been treated with elimination diets, injections, desensitization, catalysts and so on, all to little purpose. However, it was to some purpose because for a few weeks or months at the commencement of any new form of treatment her condition improved. This transient improvement is frequently seen in these cases, and it is to my mind associated with the fact that the child has become worse and the parent more solicitious; reliance of both is placed on a confident third party, and improvement results. However, before long both child and parent resume their usual relationship and back comes the asthma.

Betty's mother said that the child had no asthma until the age of three. She had always been a forward, active, restless and excitable child, and although she was always very independent and wanted to do everything for herself, she was actually nervous and inclined to cling. When it was suggested to the mother that perhaps she herself might have been dominating the child she reluctantly admitted it, but said that she was never harsh. She also admitted to being over-anxious, over-protective and perhaps a "fuss pot". She said that Betty was always very forward and independent and wanted to do everything for herself to the extent of alarming her, and she, the mother, had to restrict her. At about the time the asthma developed the child became shockingly difficult and "I had to drive her to do everything. She used to want to do everything for herself and now she won't do anything. It's Betty, do this, Betty, do that, and Betty, do the other thing. Have you washed yourself, Betty, have you brushed your teeth, Betty, have you cleaned your shoes, Betty, have you done your practice? She is so obstinate, I don't understand it!"

There was a lot of very thinly veiled antagonism between Betty and her mother. Betty told her mother one day that she was different from other mothers. This shocked her mother, but she was horrified when on another occasion she overheard Betty tell her grandmother that she hated her mother. "I don't understand Betty. I am affectionate, but not demonstrative, I can't show it, Betty is always kissed by me, she never kisses. The pleasanter side of life for Betty is bound up with honeycake, asthma and getting her own way!" The mother was always trying to dragoon the child, reasoning with her, or, as the child put it, lecturing her. She was not allowed to go to the pictures with her friends, to go to the beach with them, for fear of coming to harm, and her mother attempted to choose her friends and supervise them. At the time I saw her she was contemplating changing schools "because Betty was picking up rude things". The real reason for the child being brought to see me was that a neighbour had overheard the child say to a friend that she would not have to do such and such a thing, because if she had to do something she did not want to do she usually got asthma. I rather felt that Betty got off lightly with only asthma. In this case the mother was cooperative and Betty was a lot better. However, the

strain of self-discipline was too much for her mother and she gratefully accepted the suggestion that Betty might do better in the impersonal atmosphere of a boarding school. The asthma then cleared up with occasional mild relapses during the school holidays.

Case II.

Mrs. P., aged fifty-seven years, had suffered from asthma and hay fever practically all her life. She had many searching investigations and treatments, but continued unchecked in her asthmatic way. Quite recently she had fallen into more or less of a *status asthmaticus* and was only partially relieved by the continuous adrenaline technique. In desperation her physician, with considerable ingenuity, got her a bed in hospital. As soon as she was lifted into the ambulance her asthma subsided and she had no further attacks for the six weeks of her stay in hospital. However, they returned on her discharge to her home. When seen, she was wheezing and obviously having a good deal of expiratory distress, but during a psychological history taking her breathing rapidly became normal as her interest in her own story absorbed her. This is a common enough experience. Her story was this: She had been an intelligent, outgoing, excitable, restless, nervous sort of child. She was a good mixer, fond of the limelight, and liked doing things for herself, but she lacked confidence. Her mother was extremely dominating—in a very nice way, of course—but at the same time she was over-anxious and over-protective. When the child grew up her brothers and sisters got themselves jobs and went their ways, but someone had to stay at home with mother and obviously it would be our patient. She resented being dependent on the mother, but lacked the courage and confidence to break away as she wanted to. Then she married and almost immediately became pregnant. During her pregnancy she became ill and had numerous attacks of biliary colic and presumably cholecystitis which, because of her youth, was not diagnosed. This made her more or less of an invalid, and when the baby was born mother came to live with them to look after the child. Her ill health from recurrent attacks of cholelithiasis continued for three years, and then "the new X-ray" came in, her condition was correctly diagnosed, a gall-bladder "containing three hundred and fifty-four stones" was removed, and she regained her health. It is informative that during this period of three years of ill health and dependence she had no asthma or hay fever whatever. Three months later she developed asthma again, and has had it ever since.

The man she married was "exacting and difficult". He was an obsessional type, everything had to be just so. He was always aggressively right and would prove it by logical argument. He was extremely dominating and yet over-anxious and terrified if his wife or child were ill. He was generous with money, but had rigid and high standards and was exacting in his demands. His wife volunteered the information spontaneously that she realized very early in her marriage that if she wanted to be happy she must not argue. (Inhibition.) "Jim was always right." She also volunteered the information (and, incredible as it may seem, without insight) that the only time her husband was really nice to her was when she had asthma. In talking of her daughter, she said that she constantly impressed upon her the importance of always being nice in all circumstances, adding that "it costs you nothing". It cost her mother asthma. She related an amusing story of her husband's first attempt to teach her daughter to drive the car. He was so exacting and difficult that finally his daughter pulled the car up, got out, slammed the door, told him to drive the car himself and walked home.

Although I am not commending this behaviour, it is interesting to note that this girl does not suffer from asthma.

The reason for the present aggravation of the symptoms was that during the past twelve months her husband had at last enforced a separation from her mother. However, almost coincidently her husband had a pulmonary embolus from which he had recovered. This was followed by a second one from which he also recovered. However, this ill health had made him more exacting and difficult. It also threatened her future security.

Surely this case requires no comment? This patient went for a prolonged holiday and remained free of symptoms. She subsequently lived with her sister for some time and remained free from attacks but occasional visits to her home invariably precipitated her illness again.

Summary.

This is a highly speculative paper postulating the idea that asthmatic attacks are precipitated by disorders of cerebral inhibition in persons with the asthma diathesis; the suggestion is that this diathesis might represent an inherited autonomic nervous system stigmatization.

Conclusion.

As yet we do not know nearly as much about medicine as we think we do.

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Reports of Cases.

A CASE OF MASSIVE HÆMORRHAGE FROM PEPTIC ULCERATION IN A MECKEL'S DIVERTICULUM.

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Perth.

THE most comprehensive review of the literature on peptic ulceration occurring in Meckel's diverticulum was published by Johnston and Renner[1] in 1934. It is clear that at that time only seven cases had been reported in English journals, and no case in an Australian journal.

Before the following case is reported some general remarks may be summarized from Johnston and Renner's review. The incidence of Meckel's diverticulum is between 1% and 2% in humans. The mucosa is usually ileal in type, but in 37% (Schaetz[3]) or 48% (Hudson and Koplik[2]) it was found to be non-ileal. If it was non-ileal it was usually gastric, but it may be duodenal, jejunal or pancreatic, or a mixture of all three.

Johnston and Renner discuss 62 cases of definite peptic ulceration and 16 cases of probable peptic ulceration. Of the 62 definite cases only nine occurred in women. The complications of ulceration are hæmorrhage and perforation. Among the 62 definite cases of peptic ulceration occurring in Meckel's diverticula the following findings were recorded: (i) 26 patients presented with frank severe intestinal hæmorrhage without any suggestion of an acute abdominal emergency; (ii) 20 patients presented with acute abdominal emergencies, intestinal hæmorrhage being present but of secondary importance; and (iii) 16 patients presented with acute abdominal emergencies alone, without hæmorrhage from the intestine. Of the 62 patients 20 died.

Clinical Record.

V.D., aged forty-four years, a male patient, presented at midnight on September 4, 1945, with a history of having passed large quantities of blood by the bowel. He had been perfectly well until six hours prior to his admission to hospital, when he noticed without warning the appearance of blood from his rectum. He developed mild, colicky pain in the left side of the abdomen, both hypochondriac and iliac. The pain passed away, but he became alarmed at the amount of blood he had passed, and it was indeed this alone which had brought him to hospital.

On examination of the patient in the casualty department he was seen to be suffering from shock; he was pale and had a clammy skin, and complained of feeling faint. His temperature was 96° F., his pulse rate was 100 per minute and his respirations numbered 22 per minute. His blood pressure was 80 millimetres of mercury, systolic, and 40, diastolic. No abnormality could be found on

examination of the heart, the lungs or the abdomen. But while he was being examined he continued to pass blood from the bowel, which was bright red in colour; about one pint was lost while he was in the casualty department.

The patient was admitted to hospital, and three hours later a proctoscope was passed by the house physician, who found "extensive bleeding coming from a point three and a half inches inside the anus on the posterior rectal wall". A rectal plug was inserted, the patient was laid flat in bed and morphine was administered. His blood at this stage was described as "pure". A hæmoglobin estimation then revealed a percentage of 65, and the red cells numbered 2,800,000 per cubic millimetre. He was examined again on the morning of September 5 and apparently then left for a further twenty-four hours, as he was not losing a sufficient quantity of blood to call for comment.

On the morning of September 6 he vomited for the first time after having a cup of tea. By 10 a.m. that day he started to pass blood again and had lost one pint by 11 a.m. The rectal pack was removed and some clotted blood came away, followed by a further half-pint of dark red fluid blood. His colour became pale and his pulse rate rose to 125 per minute at 12 midday on September 6. The diagnosis was so much in doubt that the surgeon was consulted. He passed a sigmoidoscope easily into the rectum up to a distance of 25 centimetres, and dark blood could be seen gushing down from above that level. The hæmoglobin percentage was 50% at this stage, the forty-fourth hour since the hæmorrhage had started. An intravenous transfusion of blood was commenced, and in the next eighteen hours he received six pints of blood. With this and the use of morphine he appeared to settle down well, and his pulse rate and blood pressure were returned to normal limits. Some abdominal distension only was evident on examination.

At 10 a.m. on September 7, the sixty-fourth hour since the onset of hæmorrhage, his pulse rate was 112 per minute, his temperature 99·8° F. and his blood pressure 130 millimetres of merury (systolic) and 90 (diastolic). He was examined by the surgeon, who was satisfied with his condition and decided that there was no indication for immediate operation. On leaving the ward, however, the surgeon was recalled urgently, because the patient had just then commenced to lose an extremely large amount of darkish blood. Hæmorrhage therefore had not stopped, and a laparotomy was decided upon. The diagnosis at this stage was still in doubt. Hæmorrhage from the rectum itself had been excluded by the sigmoidoscopic examination on the previous day. It had been considered that an inflammatory condition of the bowel could not be responsible for so severe a hæmorrhage as had occurred, although severe hæmorrhage as an isolated symptom had been seen in a case of amœbic dysentery at this hospital some time earler. The other alternatives appeared to be a massive hæmorrhage from a neoplasm of the large bowel or from a duodenal ulcer. The latter was considered the most probable diagnosis before operation, in spite of the absence of any previous history suggesting duodenal ulceration.

Operation was undertaken at 12 midday on September 7, sixty-six hours after the onset of the hæmorrhage. A blood transfusion (the eighth pint), was begun. An intercostal nerve block with procaine was used, supplemented by local infiltration anæsthesia and by trichlorethylene general anæsthesia. An upper right paramedian incision was made and Devine's retractor was applied. The bowel was then systematically examined. The stomach and duodenum were found to be innocent. The large bowel, apart from containing dark blood, was normal. Without hesitation the surgeon then searched for a Meckel's diverticulum. A large diverticulum, fully three and a half inches long and over one inch in width at its base, was discovered and found to be distended with blood. This was ligated and divided at its base, and the stump was inverted and held in by a purse-string suture. The abdomen was closed in layers without drainage.

After operation a further transfusion of a final pint of blood was given, making the patient's ninth pint, and the fluid, given by intravenous drip, was then changed to normal saline solution. Convalescence was uneventful except for some slight degree of abdominal distension and vomiting, controlled adequately in two days by gastric aspiration and fluid given intravenously. An enema given on the third day after operation resulted in a fluid stool in which melena was obvious. After this the bowels were open rather loosely about four times a day, but after three days all traces of blood had disappeared.

The patient was discharged from hospital on the fifteenth day after operation. I saw him again a few weeks ago, ten months after operation, and a fitter person one could not meet.

Macroscopic examination of the specimen, in a good light, revealed a shallow erosion about a quarter of an inch from the tip. (Ulceration in a Meckel's diverticulum usually occurs at the base.) The open end of a small artery, which admitted a pin's head, was detected in the crater. Microscopic examination showed the mucosa to be gastric in type. The muscularis was converted into irregular connective tissue.

Comment.

A case is reported of severe hæmorrhage from peptic ulceration occurring in a Meckel's diverticulum. It is not possible to diagnose the condition in the absence of operation or autopsy; but the appearance of the blood passed, which was darker than that usually seen from a neoplasm of the colon, yet infinitely brighter than the tarry stool which occurs in hæmorrhage from a duodenal ulcer, should make one consider the possibility of hæmorrhage from a Meckel's diverticulum. Appreciation of this possibility in the case recorded resulted in the origin of the hæmorrhage being rapidly disclosed in a patient unfit for a lengthy exploratory operation.

Acknowledgements.

My thanks are due to Dr. R. R. Anderson, Acting Medical Superintendent of the Perth Hospital, and to Dr. J. P. Ainslie for permission and encouragement to publish this case.

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TWO OBSCURE CASES OF ASCENDING PARALYSIS.

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Adelaide.

DURING the recent minor epidemic of infantile paralysis, an unusually large proportion of abortive and atypical cases has been reported. These include afebrile types, types in which cerebral function alone seems to have been impaired, types in which repeated examination of the cerebro-spinal fluid has revealed no abnormality, and types in which complete recovery occurred after paralysis of considerable extent and duration. There is no doubt that many of these cases were not caused by the virus of *poliomyelitis anterior acuta*. Some, indeed, were probably non-infective in origin.

The following two patients, admitted to the Metropolitan *Infectious Diseases Hospital, Adelaide, in July, 1946*, were provisionally diagnosed as suffering from *poliomyelitis anterior acuta*. These cases are examples of the ascending paralysis syndrome, and possess certain clinical and laboratory features in common. Ascending paralysis constitutes a syndrome in the true sense. Sometimes it is caused by the virus of *poliomyelitis anterior acuta*. Sometimes it is definitely and purely toxic in origin. When

the cases with an obviously infective or toxic origin have been considered, one is left with a residue of cases which have been united under the name "Guillain-Barré syndrome". The latter entity, if entity it be, is characterized by a reversible loss of sensory and motor function, and by an albumino-cytological dissociation of the cerebro-spinal fluid. The sensory loss comes first and is accompanied by tenderness of and sometimes by pain in the affected limbs.

The following cases resemble in most but not all respects the Guillain-Barré syndrome. One patient came to autopsy and the nervous system was found to be normal on macroscopic and microscopic examination, while the other patient survived and made an unexpected recovery which began on the twenty-second day of his illness. Both patients were afebrile, and the only abnormality displayed by the cerebro-spinal fluid was an increase in the protein content. There was no impairment of sensation, and inquiries made concerning the previous occurrence of diphtheria and of the other known ætiological agents of peripheral neuritis were fruitless. There was no evidence of insect bites, such as one would expect to find in cases of tick paralysis. Cases of the last-mentioned disease have not yet been reported in Adelaide. No definite tenderness was present in the affected limbs. In both cases there was a history of mild upper respiratory tract infection about a fortnight prior to the onset of the paralysis.

One is forced to assume, therefore, that these were either cases of toxic polyneuritis, in which the motor neurones alone were affected, or examples of a hitherto unrecognized infective process which does not cause the well-marked neuronal disintegration and inflammatory reaction so characteristic of *poliomyelitis anterior acuta*.

Attempts to isolate a virus from cases of the Guillain-Barré syndrome have so far been unsuccessful. It is hoped, however, that as the syndrome becomes more widely recognized, laboratory tests may be devised to differentiate it from *poliomyelitis anterior acuta*. At the present time its 80% favourable prognosis justifies its always being included in the differential diagnosis of a case of ascending paralysis.

Case I.

R.W.M. was a male patient, aged fourteen years. Two days prior to his admission to hospital, he complained of tiredness of the legs when walking up a hill. The next day his legs were so weak that he could not stand up. On his admission to hospital on the following day, he was found to have complete lower motor neurone paralysis of both legs and both hands, and moderate paresis of the muscles acting on the elbow and shoulder joints. Slight intercostal paresis was present. Lumbar puncture revealed the pressure of the cerebro-spinal fluid to be 140 millimetres in the horizontal position, and jugular compression produced a normal response. There was a slight increase in the protein content of the cerebro-spinal fluid, but less than 20 leucocytes per cubic millimetre were found. There were no symptoms of meningeal irritation. On the following day slight neck stiffness was present, and pain was felt on full flexion of the legs. Transient urinary retention developed, and the patient's bladder had to be catheterized once. Subsequently the disease process gradually extended, so that the intercostal muscles, the muscles of the arms, and the diaphragm became paralysed in that order. On the sixth day of the disease the patient had to be put in the respirator. In spite of this he became more and more cyanosed, and gradually lost the powers of speech and deglutition. He was mildly euphoric, and remained in full possession of his mental faculties until the evening of the ninth day, when he became moribund. He died on the tenth day.

The nervous system was examined by Dr. Charles Swan, who found no macroscopic or microscopic abnormality in the central nervous system or the peripheral nerves. The changes caused by the virus of *poliomyelitis anterior acuta* were entirely lacking. The cadaver showed great muscular wasting.

Case II.

W. McG. was a male patient, aged fourteen years. Two days prior to his admission to hospital, his legs felt weak. On his admission he was found to have complete paralysis of the right leg and great weakness of both arms and the left leg. Next day both legs were completely paralysed, and great weakness of both arms and of both sterno-mastoid muscles was present. There was slight weakness of the diaphragm and intercostal muscles. Moderate stiffness of the neck was present and a complaint of pain in the back was made. At this stage the only abnormality in the cerebro-spinal fluid was a slight increase in the protein content. Seven days later, the protein content had increased so that a dense ring was formed in the absolute alcohol stratification test. For twenty-two days the clinical picture remained unchanged, apart from a transient rise of the temperature to 100·5° F. on the tenth day. On the twenty-second day a considerable increase in power was found in the arms, and the patient was able to move his legs for the first time. Since then the patient's condition has continued to improve, so that now, after three months, only slight weakness of the dorsiflexors of the feet and of the fingers remains.

Acknowledgement.

I wish to thank Dr. Alan Finger, Medical Superintendent of the Metropolitan Infectious Diseases Hospital, Northfield, for permission to publish this report.

Reviews.

PATIENTS AND APPENDICITIS.

ENGLISH's book is a somewhat philosophical discussion of patients who undergo appendicectomy, of their preparation for the operation and its surrounding circumstances, and of their investigation and post-operative treatment.

It will be of particular interest to medical students, as it begins where the student should begin—with the patient. The chapter on "Patients" is alone worth an entire book. Here the author describes each type of patient in detail: business men who have allowed their physical power and reserve to drop, and who have a lowered reserve power against emergency; the various members of a family, the fathers, the mothers *et cetera*. Of daughters he states that they are "the most easy and natural group with whom we have to deal; they are usually self-possessed and have no hesitation in telling us what they like and dislike".

He states that the medical man who deals with his patients with sympathy and understanding will have it said of him: "They came to him as patients and remained with him as friends."

Throughout the whole book the human side of the patient with appendicitis is kept well to the fore, but the actual technique of appendicectomy is rather sketchily illustrated.

OPHTHALMOLOGY IN THE WAR YEARS.

To review a work of the magnitude of "Ophthalmology in the War Years (1940-1943)", of which Meyer Wiener is Editor-in-Chief, is a difficult task.[2] In its twelve hundred pages is abstracted the world ophthalmic literature from 1940 to 1943, and the second volume, covering the years 1944 to 1946, is expected shortly. This colossal task is the result of a suggestion of British ophthalmologists to the American Academy of Ophthalmology and Oto-Laryngology in 1943. It was felt that men in the services would not have the literature available to them, and that those at home would be so busy that they would be unable to read anything worth while even if they could obtain the article. When this suggestion was made Wiener promised to cover the field. This he has done with the help of his co-editors in a surprising manner, considering that so many of these men were actively engaged in the services or doing double duty on the home front.

It appears that little of the current literature has not been dealt with; articles by Australian ophthalmologists are

[1] "Patients and Appendicitis", by Sir Crisp English, K.C.M.G., *F.R.C.S.*; *1946*. *London*: *J. and A.* Churchill Limited. 8½" × 5½", pp. 162, with illustrations. Price: 10s. 6d.

[2] "Ophthalmology in the War Years", edited by Meyer Wiener. *M.D.*; *Volume I (1940-1943)*; 1946. Chicago: The Year Book Publishers, Inc. 9½" × 6¾", pp. 1176.

included. As co-editors Meyer Wiener has had a host of helpers too numerous to mention.

It is suggested that the task set by our American colleagues should not be allowed to lapse when the abstract of the literature of the war years has been completed, but that this should be a permanent feature of ophthalmological literature in the post-war years and should appear at least at regular six-monthly intervals. It is apparent that busy ophthalmologists would welcome a continuance of this work. It would keep them conversant of current thought and the advance of the science of ophthalmology in every necessary direction.

This work can be warmly recommended to Australian ophthalmologists who have, for the past seven years, been cut off from intimate contact with ophthalmologists overseas, and who may be prevented by economic circumstances from undertaking further overseas travel in the immediate future.

STERILITY.

"PROCEEDINGS OF THE CONFERENCE ON DIAGNOSIS IN STERILITY", edited by Earl T. Engle, is a report of the conference held in New York in January, 1945.[1] As well as the set addresses, the discussions following them are included, and at the end of the book a very instructive summary of the proceedings by one of the members is given. All the various aspects of the subject are covered and the address on "History Taking for the Infertile Couple" is excellent. In this the speaker emphasizes the necessity for full cooperation of both marriage partners in enabling an accurate diagnosis to be made, and he states that "causative or contributory factors occurred simultaneously in husband and wife in 28 per cent." of cases. Another interesting address is on the subject of the testicular biopsy in which the speaker advises that biopsies from both testes should always be taken when these glands are thought to be at fault. Dr. Rubin spoke at length in the discussion on the subject of tubal patency tests, and he wisely advised that lipiodol should not be used as a contrast medium in such tests, owing to its very slow absorbance rate and the possibility that it may increase any damage already present. Mention cannot be made of all the topics that were discussed at the conference, but a complete survey of the subject is given and the book will be of practical value to all medical men whose work brings them face to face with the vexing problem of sterility.

TWO BOOKS ON THE RORSCHACH TEST.

THE purpose of Beck's second volume on the Rorschach test is given by the subheading "A Variety of Personality Pictures".[2] The first volume gave a detailed account of the nature and the scoring of the test.

In this book the author describes the personality pictures associated with high, middle and low intelligence, the problems of adolescence, schizophrenia, neurotic struggles and the effects of treatment on personality tests.

In each example the test summary, the conclusions that can be drawn about the personality from the information given in the test, the test responses and the clinical history, are given in that order. Many readers might prefer the test responses to be set out first, and for them to be followed by the test scoring, the discussion and the clinical history in that order. The demarcation of each case record could be more clearly defined.

The author discusses in great detail the information that can be gained from the test material. In his hands it is obvious that the test can give much information. How valuable this information is to the clinician remains to be shown. Some help to this end would be given if more detailed clinical histories were cited. A clinical history condensed to half a dozen lines is not very helpful. This aspect needs stressing because of the tendency to place too much reliance on special tests rather than on their use in conjunction with the ordinary clinical history and observations on the patient.

As this book is more than an introduction to the test, it will be of most value to psychologists using the test or fully conversant with it. They will welcome the interpretation of the test material. Such a book will appeal to general psychiatrists only when the author or a psychiatric collaborator indicates in which personalities or psychiatric reactions the test offers help in the understanding of the person and in the management of his disorder.

In the revised edition of "The Clinical Application of the Rorschach Test" the description of the test and how to interpret it is practically unchanged.[1] A previous criticism that a reproduction of the test blots should have been included still holds.

The second section of the book, devoted to a description of the results of the test in the various psychiatric disorders, has been enlarged by 100 pages. This added material comprises the main alteration in this edition. It includes records of persons with behaviour problems, suffering from alcoholism and epilepsy, as well as further examples of schizophrenia and the neurotic disorders.

This section is still very weak. The results of the tests are satisfactorily recorded and their interpretation is reasonable. The clinical records are too short to allow one to assess the clinical problem or to form any conclusion as to the value of the test in each case. The number of cases selected to illustrate this section could have been reduced without detraction from its value. For instance, an isolated illustration of epilepsy or alcoholism does not help in the understanding of the many personality types underlying these symptom complexes.

This book would have served its purpose as a useful introduction to the Rorschach test more satisfactorily had the size remained as in the first edition and had the examples chosen to illustrate the test been fewer and more carefully selected.

Notes on Books, Current Journals and New Appliances.

A WAR CORRESPONDENT IN THE UNITED STATES OF AMERICA.

MOST war correspondents write books; most of their books have a purpose—they are meant to instruct the reader on international affairs, or to interpret current events to him, or to predict the future. George H. Johnston, on the contrary, is a war correspondent who has written a book the keynote of which is bewilderment.[2] By a peculiar trick of fate Mr. Johnston was, as he puts it, "pulled out of the war, dunked in New York's incredibility for six months, and then lifted out, dripping and stunned, and tossed back into the war again" (the book was completed early in 1944). The first part of his stay was spent in a whirl of festivity, not unconnected with inebriety. Then he pulled himself out of the whirl, and tried to understand the place where he was and the people with whom he came in contact. But he made no attempt to interpret anything; he was content to absorb impressions, and if possible, to digest them. The reader is swung from one giddy height to another; he meets a remarkable collection of people, most of whom are likeable (we call to mind with pleasure Mr. Clinton Chesterfield Couch, whose brother's name was Jedidiah). And yet, in spite of digressions, irrelevancies and all the absence of literary style which the author has purposely used, the reader closes the book with the feeling that he has been amused and subtly given a sympathetic and helpful picture of his American brothers, which may well serve to increase his understanding of them. The book is illustrated by Sergeant Jack Hanna, of the Australian Imperial Force, with piquant (not to say saucy) drawings, from most of which the author peeps forth with a deceptively "Simple Simon" expression. This book offers good entertainment and much unconventional information.

[1] "Proceedings: Conference on Diagnosis in Sterility", sponsored by the National Committee on Maternal Health, January 26-27, 1945, New York City; edited by Earl T. Engle; 1946. Springfield, Illinois: Charles C. Thomas. 9" × 5¾", pp. 250, with illustrations. Price: $5.00.

[2] "Rorschach's Test: II. A Variety of Personality Pictures", by Samuel J. Beck, Ph.D., with a foreword by Roy R. Grinker, Lieutenant-Colonel, M.C.; 1945. New York: Grune and Stratton, Incorporated. 9" × 6", pp. 414. Price: $5.00.

[1] "The Clinical Application of the Rorschach Test", by Ruth Bochner, M.A., and Florence Halpern, M.A.; Second Edition; 1945. New York: Grune and Stratton, Incorporated. 8½" × 5¾", pp. 345. Price: $4.00.

[2] "Skyscrapers in the Mist", by George H. Johnston, illustrated by Jack Hanna; 1946. Sydney, London: Angus and Robertson Limited. 8½" × 5½", pp. 232, with many illustrations. Price: 10s. 6d.

The Medical Journal of Australia

SATURDAY, FEBRUARY 1, 1947.

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OPTIMISM AND REASSURANCE IN MEDICINE.

WHEN Solomon wrote that "a merry heart doeth good like a medicine" he wrote something that might with advantage be inscribed over the introduction to every book on medical therapeutics. Of the mental component in an illness much has in latter days been written and more possibly has been said; indeed to some persons the subject has possibly become wearisome so that they hold reference to it to be overdone. If a saying has become trite, people are apt to take it for granted with no thought of what it really means. This being so, the merry heart that is like a medicine may well be the subject of a moment's inquiry. The first question will probably be "What is meant by a merry heart?" Is it a state of effervescent exuberance in whose presence the owner of the heart in modern parlance makes "whoopee" or in times very long ago would have made the Psalmist inquire as he did of the mountains—what ailed them that they skipped like rams and the little hills like young sheep? Or is it something much more sedate—an inward cheerfulness that finds expression in action, a cheerfulness based on knowledge and born of faith and hope? Surely all grades between these two may be included. As different kinds of medicine find their right use in different ailments, slight or severe, so will the different grades of cheerfulness between the effervescent and the sedate come to man's aid. It will be said, of course, that the merry heart must be that of the man who is ill rather than that of his doctor. This is true enough, but the merry heart is required by the doctor just as much as by the patient. The doctor often has the task of bringing the sick man to his happy state and he cannot do this unless he has deeply ingrained in himself all the attributes essential to its unfolding. That the doctor must use skill in the display of his own merry qualities is obvious. He would look a little odd skipping like a ram or a young sheep, either literally or metaphorically, if he wanted to turn a sick man's thought from his suffering body to a hopeful future free from pain, however certain he might be that the coming days were sure to be bright. Of the doctor it may be said that if he wishes to encourage and reassure his patient, as he should always try to do, he must be gifted with optimism, tried and fortified.

Discussion of this subject has been suggested by the recent publication of two papers. T. G. Armstrong writes on the basis of his experience as the officer in charge of the medical division of a general hospital[1] on the use of reassurance and W. D. Stroud spoke on optimism in medicine in his chairman's address[2] to the Section on Internal Medicine at the annual meeting of the American Medical Association in July, 1946. Both papers present views that are worthy of consideration. Since optimism is the basis of reassurance, Stroud's paper should be dealt with first. However, he is concerned more with cardiac conditions, a subject on which, having been president of the American Heart Association, he is well qualified to speak. Armstrong deals with more general conditions; he has been treating young and active patients in the services, whose "main desire is to get well", and he refers in contrast to older folk who often "enjoy ill health" and to whom a disservice is rendered if their ailments are cured. If in his enthusiasm Armstrong finds the matter simpler than it really is, we may forgive him for his insistence on several points. "No patient can have full confidence in his doctor if a complete examination has not been carried out, and no doctor can confidently reassure his patient without having carried out such an examination." Many a practitioner early in his career resolves never to embark on a course of treatment without making an exhaustive study of the patient; later on he grows tired, or he imagines he can see without looking, since he has seen so many patients who seem to be just like the one before him. But all patients are not easily deceived and reassurance from such a man must fall flat. Again, Armstrong holds that organic disease should be diagnosed only when there is reasonable proof that it is present. By reassurance Armstrong means the allaying of a patient's anxiety, and he points out that it is of value not only in treating a patient with a neurosis but also in the treatment of one with organic disease. It is not uncommon to find that a patient who is in great fear of an organic disease becomes equable and almost serene in his outlook when he finds that he is grievously afflicted and has to face a grim future. Everyone will not agree with Armstrong that too few patients are told the cause of their complaint and that silence, except in the gravest maladies or with patients of the lowest intelligence, is inconsiderate and even dangerous. The important point to be considered is whether it will benefit the patient to be told all about his ailment; if not, then he should be told only so much as will help him in his recovery or in his leading of a placid existence if he cannot recover. The one proviso, of course, must always be remembered—that if an intelligent patient wants to know all the facts, he should be told. To make decisions in all these circumstances is not easy and the making of a decision in some instances calls for many years of experience. It is clear then that from time to time reassurance cannot be given, but that encouragement and the bolstering of morale must

[1] *The Lancet*, October 5, 1946.

[2] *The Journal of the American Medical Association*, October 19, 1946.

take its place. Armstrong thinks that if a functional disturbance is diagnosed the medical attendant should appreciate his good fortune rather than regard the patient's condition as something in which he should lose interest. Unfortunately all doctors are not quite at home in the treatment of functional ailments, and some stand in need of encouragement and the bolstering of morale themselves when faced with these conditions. Armstrong thinks that in the matter of reassurance it is useful to know the patient's own diagnosis of his condition and also what diagnoses other doctors have made in respect of it. Whether this is necessary will generally become clear as investigation of the patient's condition proceeds. A last point which needs elucidation is the occurrence of any emotional upset at or before the onset of the illness. We finally agree that adjuvant treatment should be short in order to avoid the suggestion that the patient has a serious disease. This may perhaps be looked on as reassurance by suggestion, an important point since most of the reassurance given by a medical practitioner is of this kind rather than that given deliberately in a set programme by word of mouth.

Turning now to Stroud's paper, we find him pleading for less restriction of patients with cardiac and vascular disabilities. It is unfortunately true that many children have been made miserable and their parents unhappy by physicians' serious attitudes to unimportant heart murmurs. Stroud thinks that more have been made invalids in this way than by rheumatic heart disease—an unpleasant thought. He also has something to say about the misuse of the sphygmomanometer and the inability of some practitioners to interpret electrocardiographic tracings. He believes—and others are with him in the belief—that too many doctors urge patients to give up their employment and become invalids on minor electrocardiographic findings. There is no doubt that a label of myocardial degeneration affixed to a patient will make him introspective and apprehensive. The greatest care is necessary in regard to what a patient is told about his heart. Stroud is often astounded at how well patients do who have had non-fatal coronary occlusion with myocardial infarction. Two of the most important forms of treatment in acute myocardial infarction are rest and reassurance. Fear probably accentuates myocardial irritability through the effect of epinephrine. Stroud would have the thanks of thousands of invalids if he could make their medical attendants believe with him that "in most cases it is safe to allow a patient to use a commode with much less strain on the circulation than using a bed pan". Stroud believes that too many patients after coronary occlusion are made invalids unnecessarily. It is his experience that after adequate circulation has developed it really does not seem to matter what activities the patients carry on within reason from the standpoint of the development of further coronary occlusions. Such views as this may be adapted to patients suffering from other types of condition.

In this discussion the merry heart with which we started has been interpreted in the widest sense. The reference has been chiefly to what the medical attendant should or should not do. He will do or will not do these things according to the degree of intelligent optimism that he has—according to the merriness of his own heart. And by and large his own merriness sets the tempo for his patient—there be some patients fortunately whose inherent merriness of heart is spontaneous and unquenchable and they are among the greater joys of practice. Having started with words of Solomon that a merry heart doeth good like a medicine, we do well to remind ourselves how important it is to pursue that goal. If we fail we shall have as our guerdon a broken spirit and this Solomon tells us "drieth the bones".

PYROSIS.

THE term "pyrosis" is usually defined as the equivalent of heartburn, and inquirers are enlightened by being informed that this means a burning sensation felt behind the sternum, associated with a regurgitation of acid fluid from the stomach. A clinical analysis of such a symptom cannot be very easy to make, for it is hard to decide just how rigid a definition should be adopted, and it is still more difficult to get at what is in the mind of many patients, who have a winning habit of crediting their doctors with the gift of mind reading. An interesting analysis of this symptom has been made by G. W. H. Schepers, of Johannesburg, who essays to determine its cause and significance.[1] He remarks that the social significance of dyspepsia is greater than that of carcinoma of the digestive tract. This is no doubt true, and the remark also indicates that the author has only included in his analysis those cases in which he has been satisfied that the symptom arose from some disturbance of the digestive organs, whether direct or reflex. He makes a further statement which perhaps might interest the politically minded among doctors, that a dictator with dyspepsia is probably a more dangerous epiphenomenon of civilization than one with a carcinoma. It is at once evident that Schepers is concerned in part at least with a consideration of the relation of personality with this particular symptom.

He includes in his definition of pyrosis an oppressive burning sensation below the xiphisternum, associated with acid eructations and flatulence, with also possible extension of pain to the inferior angle of the scapula. The explanation of it is not very satisfactory, especially as the association with hyperacidity, so generally considered to be common, is by no means always established by investigation. It is of course well known that antacids may give symptomatic relief even to those dyspeptics who have complete achlorhydria. Schepers has divided the associated clinical features in his cases into various categories. He has found that age makes little difference, that most of the patients were sthenic in type and were overweight, that the symptom was most troublesome between meals and was often associated with flatulence, and that a definite regurgitation of acid fluid was not always noticed. It was unusual to find undoubted evidence of organic disease of the digestive tract; in fact, radiological investigation revealed signs of ulcer in only 7% of cases, though irritable duodenum was found in 33% and affection of the gall-bladder and the appendix was rare. Moderate anæmia was found in 22% of cases. It was thought that the proportion of cases in which infection of the upper part of the urinary tract was present was unexpectedly high, and obstruction of the lower part of the tract was also observed in a significant number. Insufficient information is given by the author to enable the reader to be sure that the relationship is pathologically or statistically valid. Some enlargement of the thyreoid gland was found in over 80% of cases, and in two-thirds of the total the basal metabolic rate was significantly lowered. Nervous symptoms, as might be expected, were common. One interesting observation is that paræsthesiæ were reported by 95% of the patients; these were referred to the trunk, hands and face. In fact the author regards pyrosis as a visceral form of paræsthesia, the causal irritation arising in the upper part of the digestive tract, and giving rise to sensation in the territory of the seventh, eighth and ninth thoracic

[1] *The American Journal of Digestive Diseases*, October, 1946.

somatic nerves, the earlier links in the chain being the autonomic nervous connexions. He is also prepared to believe that the original irritation may arise outside the digestive tract, for example, in the renal tract. He states that treatment which relieves the pryosis will also relieve the associated paræsthesiæ.

Sensory phenomena are notoriously difficult to assess. Even the effort to understand the patients' descriptions may unduly emphasize their importance, and measurement, so valuable in all scientific work provided it rests on sure foundation, is often impossible. But qualitative inquiry is of value in many cases, for if it can be established that a given symptom exists; whether this narrows or broadens the field of investigation, it may help in the end to arrive at the correct answer.

CORONARY OCCLUSION IN YOUNG ADULTS.

In August, 1944, attention was drawn in these columns to some work by A. J. French and W. Dock on fatal coronary arteriosclerosis in young soldiers.[1] The eighty fatal cases reported by them occurred among soldiers aged from twenty to thirty-six years. The series had many interesting features. Thus in 39 of the 80 cases areas of fibrosis were noted in the heart, but in only six of these cases had the soldier complained before the fatal seizure of symptoms that might have been related to the heart; overweight was a prominent feature and arteriosclerosis the underlying lesion. It was concluded that in all probability the actual incidence of coronary disease in the age group under consideration was much more frequent than was suggested by clinically unrecognizable and fatal cases. Sir Maurice Cassidy in the Harveian Oration of 1946[2] states that it has become evident that coronary disease in young subjects is not so rare as used to be thought. He refers to the findings of French and Dock and also to others by Maurice Newman which will be mentioned presently. He discusses the histories of 1,000 patients of his own and points out that about 70% of all patients were between fifty and seventy years of age. In 26 males the age at onset was under forty and in two it was under thirty years, the youngest being twenty-six. Only three of the affected women were under forty years of age and none were under thirty. Newman's observations[3] to which we wish to draw special attention cover fifty cases collected by him in the Ministry of Pensions, Great Britain (he is principal medical officer), from records of service men and women who had been invalided with or died from coronary occlusion or thrombosis. The fifty persons were aged up to thirty-five years. The youngest patient was twenty years of age and 22 of the 50 patients were in their twenties. The diagnosis of coronary thrombosis was established clinically in 11 cases and by necropsy in 39. Unfortunately no information regarding family history, ancestral longevity and habits including the use of tobacco was available. On the other hand every subject was examined on entry into the services and in many cases several times subsequently. The striking feature was the good physical development and nourishment of the subjects. In view of the observation of overweight by French and Dock in their series, it is of interest to note that in 21 of the 39 necropsies in Newman's series remarks such as "well nourished", "powerfully built" and "obese" were made. Of the 50 subjects 45 were graded fit on entry. Even in the five exceptions the lowering of the category was due to some slight disability entirely unconnected with the general physical development. The heart was regarded as normal in every case. There were six cases in which previous infection probably had an ætiological significance—three persons gave a history of rheumatic fever, two gave a history of scarlet fever, and one had suffered from chronic suppuration. Among the 50 cases there were 39 deaths. Further, 33 of the 39 persons who died were found dead or died almost immediately after a collapse. Practically all the subjects who died suddenly were apparently fit and healthy men who had previously carried out their military duties without any sign of cardiac distress, the disease being unsuspected during life and diagnosed only at autopsy. In contrast to the high mortality in his series, Newman quotes a recording of Conner and Holt of an immediate mortality of 16·2% among 287 patients of all ages; he also quotes a statement by W. Boyd that less than 25% of patients die in the first attack. The latency of coronary disease until the occurrence of sudden death is emphasized by Cassidy, who records a statement of Professor Hume, of Newcastle, that since 1911 he has performed or attended post-mortem examinations on 160 miners who had died suddenly and unexpectedly in the pit or in close proximity to it. The cause of death in each instance was coronary atheroma, and most of the men had been working regularly without complaint up to the moment of their fatal collapse. Only 40 had premonitory symptoms, and in about half of the 160 cases there were old fibrotic scars in the heart muscle. It is important to note that in 37 of the 39 cases in Newman's series in which autopsy was performed, the degenerative atheromatous changes usually found in the coronary disease of older subjects were present; in 29 cases there was no thrombus.

Apart from the clinical interest in Newman's series there is a clear indication of the need for further research in this subject of coronary disease in young adults. Cassidy is at great pains to show that even during so short a period as the last twenty years, the increased prevalence of coronary disease is beyond question. At the same time he states that figures from the Metropolitan Life Insurance Company of New York show that there is a lessened mortality from diseases of the heart, arteries and kidneys during the last thirty years and that this affects chiefly persons up to the age of twenty-five years. This he thinks is attributable to more efficient treatment of the acute infections which are largely responsible for cardio-vascular and renal deaths in the lower age groups. Any light that is shed on coronary disease in young adults will almost certainly help to elucidate the problem of coronary disease as it affects persons of all ages.

[1] *The Journal of the American Medical Association*, April 29, 1944.
[2] *The Lancet*, October 26, 1946.
[3] *The Lancet*, September 21, 1946.

BENIGN LYMPHOCYTIC MENINGITIS AND GLANDULAR FEVER.

Glandular fever was described first by Pfeiffer in 1889 as an acute infectious disease characterized by painless swelling of the cervical glands and slight constitutional symptoms. Benign lymphocytic meningitis was described by Wallgren in 1925 and called "acute aseptic meningitis". At first glance there appears to be nothing to associate these two disorders. But, as Henry Tidy points out, our conception of each of them has altered considerably.[1] For instance, "benign lymphocytic meningitis has become linked with certain cases of meningoencephalitis, of encephalitis, of lesions of the cord, and lesions of the peripheral nerves". Glandular fever has been found to be a much less simple disease than the one described by Pfeiffer. First, mononucleosis was noted. Later it was discovered that at the onset polymorphonuclear leucocytosis might be present. An important discovery was the existence of heterophile antibodies to sheep's red corpuscles (the basis of the Paul-Bunnel test). In some cases a severe febrile disturbance of longer or shorter duration occurs before glandular enlargement and before the appearance of heterophile antibodies. Tidy points out also that lesions of the central nervous system may occur in glandular fever before, during or after the appearance of glandular enlargement. Meningitis, meningoencephalitis, encephalitis, or paralysis of cranial or peripheral nerves may occur. In these cases mononucleosis and a positive reaction to the Paul-Bunnell test are found. The changes in the cerebrospinal fluid are identical with those in benign lymphocytic meningitis. In a search of the literature Tidy has been unable to find any reference to glandular enlargement in benign lymphocytic meningitis or any evidence to show that search was made for glandular enlargement. It is possible therefore that all the cases of benign lymphocytic meningitis on record are cases of glandular fever.

[1] *The Lancet*, December 7, 1946.

Abstracts from Medical Literature.

DERMATOLOGY.

Epithelioma of the Skin.

JOSEPH A. ELLIOTT AND DAVID G. WELTON (*Archives of Dermatology and Syphilology*, April, 1946) hold that the proper treatment of epithelioma of the skin is still one of the most important responsibilities of the dermatologist. Epithelioma improperly treated can be one of the most dangerous of all diseases of the skin, yet there is no cutaneous disease which responds more readily to proper therapy and yields as uniformly good results as skin cancer in its early stages. The subject material of the authors' report is drawn from the private practice of one of them over the twenty-two year period 1919 to 1941. During this time, 2,081 patients with a diagnosis of epithelioma were seen; of these, 208 patients had lesions of mucous membranes and their cases are not included in the report; 79 patients failed to return for treatment, and 39 were referred for surgical treatment or high voltage X-ray therapy. Cases of Bowen's disease, Paget's disease and malignant melanoma are excluded. There remain 1,742 patients with a total of 1,928 epitheliomata. There is a certain type of skin which is more fertile soil for the development of epithelioma, namely, that of the person with blond, sandy or ruddy complexion which freckles readily but does not tan, usually associated with light hair and eyes. These patients apparently have low actinic tolerance, and when their occupations or avocations require prolonged exposure to the sun and wind they are prone to acquire keratoses and epitheliomata. There is likewise a general impression of some standing that epitheliomata are less likely to develop in dark-skinned persons than in light-skinned persons exposed to similar conditions. The authors quote Molesworth's statement that in Australia skin carcinoma is at least five times as common as it is in Great Britain, with a similar racial stock, and his view that this difference is due to the greater exposure to sunlight by a major portion of Australia's population. Briefly, the method of treatment employed by the authors most often is as follows: First, thorough curettage of all abnormal tissue is followed by electrocoagulation or desiccation of the new surface; then the area thus treated *plus* a peripheral margin of normal skin is exposed to 600*r* to 800*r* of unfiltered X rays on the same day. This exposure is repeated, preferably every four to seven days, until a total of 2,400*r* to 3,000*r* has been given. The authors believe that this method of administering X rays produces results superior to those obtained when the same total dose is given at one sitting. The time required for healing ranges from four to seven weeks, depending on the original size and depth of the lesion.

Immunization Therapy for Lichen Planus.

HANS BIBERSTEIN AND JACOB WACHTEL (*Archives of Dermatology and Syphilology*, April, 1946) report a number of experiments with regard to immunization for *lichen planus*. These experiments are based on the infectious theory, which is supported by certain clinical and experimental experiences, by certain therapeutic reactions and by observations which suggest contagiousness. Extending full consideration to the other hypotheses mentioned, the authors believe that the theory of infection as the cause of *lichen planus*, advanced by Hallopeau and Jadassohn, is the most probable one, and that a virus may be the cause, as suggested by Darier, Lipschutz, Klaar and Rosner, and Kogoj. The antigen for the treatment of *lichen planus* was prepared as follows from *lichen planus* tissue in which the virus was assumed to be present. Papular or hypertrophic lesions, cleansed with alcohol and ether, were anæsthetized with procaine hydrochloride and epinephrine hydrochloride by subcutaneous injection and removed with a sharp spoon or a curette. The borders between the *lichen planus* infiltration and the normal tisue of the cutis were easily felt, so that any deeper injury to the cutis was avoided. The erosions healed within ten to fourteen days with the use of a mild salve (for example, boric acid ointment or bismuth subgallate-zinc oxide paste). The scrapings were cut into small pieces with scissors and ground in a mortar with isotonic solution of sodium chloride until a pulp resulted. Approximately three parts of the solution were added to one part of solid particles. This slush was kept at room temperature for twenty-four hours and then placed into a water bath at a temperature of 56° to 60° C. for sterilization for two hours. After bacteriological sterility tests in liquid and on solid media did not show any growth, the suspension was filtered through sterile gauze in order to remove particles which would not pass through a needle. Phenol was added as a preservative (0·5% solution). The antigen thus prepared was administered intracutaneously twice a week; two injections, 0·1 millilitre each, were given at each treatment. This dosage was based on experience published in a previous paper. The immunization therapy was applied to forty patients, many of whom had previously been treated by other methods with unsatisfactory results. Seven of them discontinued treatment prematurely, so a conclusion cannot be drawn regarding them. Of the remaining 33 patients, three were not benefited, six improved considerably, eight were practically cured, retaining only insignificant remnants which cannot be explained, and sixteen were completely cured clinically. Altogether 30 of the 33 patients who could be followed up were favourably influenced. Fifteen to twenty double injections given twice a week are considered to be sufficient for treatment in the majority of cases in which there is a reaction.

Epidermal Sensitivity to Penicillin.

HELEN KELLER GOTTSCHALK AND RICHARD S. WEISS (*Archives of Dermatology and Syphilology*, April, 1946) discuss contact dermatitis with regard to penicillin. They state that contact dermatitis has been reported in persons who handle the salts of penicillin. The application of test patches to some of them has revealed that the sensitivity may be due to penicillin salts. Silvers administered patch tests to a chemist in whom contact dermatitis had developed while he was handling amorphous sodium penicillin. Patch tests elicited positive reactions to the "impure" yellow amorphous sodium penicillin and no reaction occurred to pure white crystalline sodium penicillin. Some investigators who have applied penicillin locally have reported the development of contact dermatitis during treatment. Patch tests were used for additional information by two of these investigators. Roxburgh treated 75 patients suffering from pyogenic infections of the skin with local application of penicillin. He stated that in two of his patients penicillin ointment appeared to be irritating to the skin. The authors' comment is as follows. Of seven persons who had had prolonged contact with penicillin sodium, seven were sensitive to the patch tests with penicillin ointment and none to the tests with the ointment base. Three reacted to penicillin sodium dissolved in isotonic solution of sodium chloride on test patches. These volunteers had apparently become sensitized to penicillin through their contact with it. Study of the literature revealed that persons who had worked with penicillin might become sensitized to it, and the authors think that their results confirm this observation. They conclude that penicillin when used locally is capable of producing contact reactions, sometimes severe, in an unknown percentage of the population. Penicillin, apparently, is not a primary irritant in the concentration employed in the studies reported by them. By means of patch tests they were able to produce epidermal sensitization to a penicillin ointment in 4·5% of 200 persons. They did not have enough material to determine whether epidermal sensitization predisposed to dermal or vascular sensitization. What little evidence they had indicated that this was not the case.

Diagnosis of Infestation with Sarcoptes Scabiei.

EUGENE A. HAND (*United States Naval Medical Bulletin*, June, 1946) discusses the life cycle of *Sarcoptes scabiei* var. *hominis*. He reports that, while making a scraping of the skin on an Australian army private suspected of having khaki dermatitis or tinea of the groin, the accidental finding of an adult female acarus led to the routine search for this organism in all cases of typical and atypical scabies. Friedman noted that the acarus could easily be demonstrated by the scrape and smear method and also that the diagnosis could be made from finding the eggs, filled or empty, skeletal parts of the acarus, the larvæ, and the scybala as well as from the adult acarus. Mary Hopper found this scrape and smear method also by accident and has used it for six to eight years at the Cornell University Medical School. The author's object is to help to popularize this method of making the diagnosis of scabies a more scientific matter. This method is not well known. Photomicrographs of the adult female, the hexapod larvæ, the acarus eggs in various stages of hatching, empty acarus eggs, the acarus burrow, skeletal parts of the acarus, and scybala also are reproduced by the author. With reference to the technique of finding the *Sarcoptes scabiei* and its parts, the author states that it is best to make a careful search over the parts affected to pick out lesions that will most likely give a positive diagnosis. Here experience helps a great deal. Ordinary

rules of cleanliness and asepsis should be followed. For lesions on the fingers, on the interdigital webs, on the hands, wrists and axillæ a sterile, sharp razor blade is used to shave off a very thin slice of the vesicle, burrow or cuniculus, or excoriated papule. For lesions on the abdomen, genitalia, buttocks and other parts less easily reached, scraping of the lesion with a sterile razor blade or Bard-Parker knife is best. The thin slice of epidermis, or the scraping, is then placed on a glass slide, covered with a cover slip, and mounted in 10% or 20% potassium hydroxide solution, glycerin, water or saline solution. The slide is then examined under low power; the high power is used when needed. Potassium hydroxide solution is used when a quick diagnosis is sought as it rapidly breaks up the skin cells making it easy to see the acarus or any of its parts or products. If saline solution or water is used it is possible at times to find a scabies organism near the surface of the skin examined and then evidence of vitality can be seen.

Treatment of Infected Sebaceous Cysts.

JACK FISHMAN (*United States Naval Medical Bulletin*, June, 1946) reports that in naval dispensaries many patients complain of sebaceous cysts, usually infected, and frequently located about the ears. Often they give a history of previous incisions or spontaneous drainage of these cysts with recurrences. Complete removal of the cyst wall is necessary to avoid recurrence. This, however, is often extremely difficult in the presence of infection as well as time consuming. Another method has therefore been employed in 512 cases and found to be highly satisfactory. The skin over the cyst is cleansed with tincture of "Merthiolate" or other skin antiseptic. With a sharp-pointed small sterile scalpel a stab is made into the cyst. The contents are evacuated. A small solid piece of silver nitrate, approximately 0·1 gramme, is placed into the cyst cavity through the stab wound and a sterile dressing or "Band-Aid" is applied. In about twenty-four hours the cyst wall is discoloured by the silver nitrate, easily recognized, and loosened from the surrounding tissue. It is grasped with a hand forceps and easily removed *en masse*. The cavity is then allowed to heal by granulation.

UROLOGY.

Renal Calculi and Hyperparathyreoidism.

E. N. COOK AND F. R. KEATING (*The Journal of Urology*, December, 1945) write to reemphasize the important relationship between hyperparathyreoidism and the formation of renal calculi. They contend that clinicians tend to discount or at least to overlook this connexion and they believe that hyperparathyreoidism is a relatively frequent cause of renal calculi. Albright and his associates in Massachusetts indicated clearly that renal calculi were much more frequent consequences of hyperparathyreoidism than were the osseous lesions, which, in the form of *osteitis fibrosa cystica*, were thought to be the invariable manifestation of the underlying disease. Although definite hyperparathyreoidism is seldom diagnosed in cases of renal calculi, the authors think that very careful metabolic studies are necessary to exclude altogether the possibility of some degree of this disease. In some cases reported in the literature hyperparathyreoidism has been caused by parathyreoid adenocarcinoma. To describe a characteristic sequence of symptoms of hyperparathyreoidism is very misleading, since the symptoms vary greatly. General muscular weakness, fatigue and constipation have been noted and are ascribed to decreased muscular and nervous tone, which results from hypercalcæmia. Polyuria and polydipsia are often present and may lead to a mistaken diagnosis of *diabetes insipidus*. The renal symptoms are those usually associated with calculi and there is a common tendency to recurrence. The third group of symptoms results from involvement of the skeleton. These symptoms are less common than the renal symptoms. Weakness of the legs and vague aching and pains in the bones and joints occur. Pathological fractures, cysts and tumours of bone and every variety of skeletal deformity may be encountered in cases in which the disease is severe. Repeated examinations of the blood to determine the calcium or phosphorus content may be necessary and final judgement may have to be delayed until the average of many determinations is made. If the average figure for serum calcium content is above 10·5 milligrammes per 100 millilitres, the possibility of the disease must be considered. The serum phosphorus content is usually reduced. The normal value is 3·5 milligrammes per 100 millilitres. Determination of the concentration of total serum protein is necessary because of its importance in the interpretation of equivocal values for serum calcium. If the total protein is low, a normal or even slightly reduced serum calcium content may indicate hyperparathyreoidism. The concentration of alkaline serum phosphatase is increased if associated disease of bone is present. The treatment of hyperparathyreoidism is surgical. Where there is a parathyreoid adenoma it is removed. Where there is a primary hypertrophy of all parathyreoid tissue, subtotal resection of the parathyreoid glands is advocated. At the Mayo Clinic, as a result of careful search, eighteen cases of hyperparathyreoidism were observed in eighteen months. In thirteen of these cases renal calculi were the presenting symptoms and signs of bone involvement were minimal or absent.

Variations in Semen Quality.

J. MACLEOD AND L. M. HEIM (*The Journal of Urology*, November, 1945) set out their results in the study of the metabolism of human spermatozoa in semen specimens taken over some years from healthy young normal medical students. They studied (*a*) the variation in semen quality among individuals, (*b*) the variation in quality in the individual over a period of many months, and (*c*) these data in comparison with those obtained from semen specimens in both fertile and sterile marriage. All specimens were ejaculated into a sterile glass container. They were examined within one hour and then, being kept at room temperature, at various intervals up to twenty-four hours. From certain individuals specimens were obtained twice weekly for several months. It has generally been accepted that pregnancy will not occur if the sperm count is less than 60 million per millilitre. Thus, in considering the potential fertility range of this group of 100 young men it was not reassuring to note that 12% consistently showed counts of 30 million or less, and 2% were absolutely sterile. Also in the case of most individuals with low counts, even between 60 and 80 millions, defects in morphology and motility of the specimens were prone to appear. These findings among apparently healthy young men were not appreciably different from those found in a group of married men of known fertility. From these studies also it appears that there is no seasonal variation in spermatogenesis and semen quality.

Methenamine Mandelate in Urinary Infections.

G. CARROLL AND H. N. ALLEN (*The Journal of Urology*, June, 1946) have studied a series of two hundred cases of urinary tract infection treated by mandelamine (a combination of mandelic acid and hexa-methylamine-tetramine). Each tablet contains 0·13 gramme of mandelic acid and 0·12 gramme of methenamine. Three tablets are given every six hours. The only other surgical treatment was the carrying out of any appropriate surgical measures. The best percentage of cures (85%) was obtained in *Escherichia coli* infections, moderately good results were obtained in *Staphylococcus aureus* and streptococcal infections and fairly good results in *Staphylococcus albus* and mixed infections. Toxic effects were almost completely absent. *Bacillus proteus* and *Aerobacter aerogenes* were highly resistant to this drug. Mandelamine maintains an acid urine without diet restriction or other drug therapy except with urea-splitting infecting germs when the urine remains persistently alkaline.

Genito-Urinary Tuberculosis.

J. A. LAZARUS (*The Journal of Urology*, February, 1946) has made a clinical study of sixty-three cases of urogenital tuberculosis and declares that the incidence of this disease has greatly declined in the United States of America in the past two decades. However, post-operative fistula and wound disintegration occur too frequently still after nephrectomy for tuberculosis. The author has found that a combination of deep X-ray therapy with ultra-violet irradiation has almost completely eradicated this disagreeable complication; it is also effective treatment for sinuses already present. Deep X-ray therapy is also, when applied over the bladder, most useful in alleviating pain in that organ and in promoting healing of ulcerations. Of the sixty-three cases in this study, ulcero-cavernous lesions in the kidney occurred in 50·8%, pyonephrosis in 31·8%, multiple abscesses in 8%, and lesions discovered only on section in 9%. Bladder ulceration was present in more than half of all types of renal tuberculosis. In most cases of nephrectomy, deep X-ray therapy was commenced soon after operation and usually about an average of fifteen treatments were given. A careful course of rigid hygiene, similar to that employed in pulmonary tuberculosis, is mandatory in all cases of nephrectomy for renal tuberculosis.

Bibliography of Scientific and Industrial Reports.[1]

THE RESULTS OF WAR-TIME RESEARCH.

During the war a great deal of research was carried out under the auspices of the Allied Governments. It has been decided to release for general use a large proportion of the results of this research, together with information taken with former enemy countries as a form of reparations. With this end in view, the United States Department of Commerce, through its Publication Board, is making a weekly issue of abstracts of reports in the form of a "Bibliography of Scientific and Industrial Reports". This bibliography is now being received in Australia, and relevant extracts are reproduced hereunder.

Copies of the original reports may be obtained in two ways: (a) Microfilm or photostat copies may be purchased from the United States through the Council for Scientific and Industrial Research Information Service. Those desiring to avail themselves of this service should send the Australian equivalent of the net quoted United States price to the Council for Scientific and Industrial Research Information Service, 425, St. Kilda Road, Melbourne, S.C.2, and quote the PB number, author's name, and the subject of the abstract. All other charges will be borne by the Council for Scientific and Industrial Research. (b) The following reports may be obtained in approved cases without cost on making application to the Secondary Industries Division of the Ministry of Post-War Reconstruction, Wentworth House, 203, Collins Street, Melbourne, C.1. Copies of these are available for reference in public libraries.

Further information on subjects covered in the reports and kindred subjects may be obtained by approaching the Council for Scientific and Industrial Research Information Service, the Secondary Industries Division of the Ministry of Post-War Reconstruction, or the Munitions Supply Laboratories (Technical Information Section), Maribyrnong, Victoria.

PB 1694. MISCELLANEOUS MEDICAL REPRINTS. (Microfilm.) No date. 1,036 pp. Price: Microfilm, $13.50; Photostat, $70.00.
This report consists of a microfilm roll containing articles, mostly reprints, from various German neurological and psychiatric journals. The material on this roll was found by members of the United States Army Medical Corps in the files of Dr. Hans Spatz and other members of the anatomical and general pathological section of the Kaiser Wilhelm Institute, Berlin-Buch, Germany.

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4. Welte, E.: *Zur formalen Genese der traumatischen Mydriasis. Oculomotoriuswurzelschädigung durch einseitiges Verquellen des Uncus Hippocampi* (Formal Genesis of Traumatic Mydriasis. Injury of the Root of the Oculomotor Nerve Resulting from Unilateral Protrusion of the Uncus Hippocampi), *ibidem*, 8, 217 (1943), 18 pp.

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[1] Supplied by the Information Service of the Council for Scientific and Industrial Research.

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FUHR, I., AND KRACKOW, E. H. Cyanogen chloride, LC 50, for rats: two minute exposure. (Chemical Warfare Service. T.R.L.R. 27.) Off. Pub. Bd., Report, PB 11553. 1944. 8 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Tests show that the LC 50 of cyanogen chloride for rats exposed two minutes and observed fifteen days is 10·1 mg./1. It is recommended that concentrations of cyanogen chloride be determined in field tests both by bioassay and by chemical analysis. A chart and tables present test data. For tests on rabbits, see PB 11557, under Krackow, E. H., and on goats, see PB 11552, under McGrath, F. P.

GALDSTON, MORTON. Distributing hose for field oxygen therapy. (Chemical Warfare Service, Edgewood Arsenal. Medical Research Laboratory, Report 6.) Off. Pub. Bd., Report, PB 9567. 1943. 15 pp. Price: Microfilm, 50c.; Photostat, $1.00.

The object of the work described in this report was to evaluate a field hospital oxygen therapy outfit for prevention and treatment of chemical casualties. It consists of a rubber hose with outlet valves, oxygen masks which can be attached to the valves, a flexible tube connector for attachment to two oxygen cylinders, a reducing valve, and a spindle and box to store all parts. The oxygen distributing hose when attached to a commercial oxygen cylinder can deliver at least four to eight litres of oxygen per minute to twenty subjects simultaneously. It was recommended that the subject item be standardized with the modifications described. Table and photographs are included.

GILPIN, BURHL B. The treatment of chemical casualties. III. Mustard injuries of the skin treated with normal amyl salicylate and No. 82 ointment. (Chemical Warfare Service. Medical Division. MD(EA) Memorandum Report 37.) Off. Pub. Bd., Report, PB 13669. 1942. 14 pp. Price: Microfilm, 50c.; Photostat, $1.00.

An outline of the treatment of several case reports at the Station Hospital, Edgewood Arsenal, is given. Distinction between amyl salicylates is discussed. It is recommended that normal amyl salicylate be considered for field use in the treatment of mustard burns of the skin. No. 82 ointment should be used on mustard burns of the face. Formulæ are included.

GILPIN, BURHL B., JUNIOR. The treatment of chemical casualties. IV. Mustard burns of the skin treated with iso-amyl salicylate. (Chemical Warfare Service. Medical Division. MD(EA) Memorandum Report 90.) Off. Pub. Bd., Report, PB 11387. 1943. 11 pp. Price: Microfilm, 50c.; Photostat, $1.00.

The purpose of this study was to determine by clinical trial the value of iso-amyl salicylate (3 methyl-butyl salicylate and 2 methyl-butyl salicylate) for the treatment of mustard burns of the skin, and to compare these results with those previously reported for treatment of mustard burns with normal amyl salicylate. The application of either normal amyl salicylate or iso-amyl salicylate to the intact mustard blister is believed to be the treatment of choice for mustard burns of the skin. Neither of the amyl salicylates may be used in proximity to the eyes because of their lachrymatory effects.

GINZLER, ARTHUR M., AND DAVIS, M. I. J. The pathology of mustard burns of human skin. (Chemical Warfare Service. Medical Research Laboratory. MRL(EA) Report 3.) Off. Pub. Bd., Report, PB 9564. 1943. 46 pp. Price: Microfilm, 50c.; Photostat, $4.00.

An investigation was made to study the histopathological evolution of experimentally produced mustard burns of human skin, and to correlate their clinical appearance with the microscopic changes. Detailed findings are listed in the appendix. Following the application of mustard to the skin of humans, there is an interval of latency, pathologically as well as clinically. This is followed by the development of erythema resulting from capillary dilatations within the corium, associated with vascular alterations and perivascular exudations. Irreversible injury to cells of the epidermis begins to appear as the erythema becomes manifest. This is evidenced by a vacuolar or hydropic intracellular degeneration. Coalescence of such cells and exudation of fluid result in an intraepidermal vesicle. More severe or intense injury may result in a coagulation necrosis of the epidermal cells.

GINZLER, ARTHUR M., AND GILPIN, BURHL B., JUNIOR. The toxic effects of acetylene tetrachloride on the operating personnel of the M-1 impregnating plant. (Chemical Warfare Service. Medical Division. MD(EA) Memorandum Report 105.) Off. Pub. Bd., Report, PB 11445. 1943. 20 pp. Price: Microfilm, 50c.; Photostat, $2.00.

Of two platoons, divided into three shifts each, which operated the M-1 impregnating plant for a total of 1,000 hours, only one man showed clinical or laboratory evidence of acetylene tetrachloride intoxication. This man, after a completion of about two-thirds of the 1,000 hour project, showed laboratory evidence of liver damage followed by progressive jaundice as a result of acetylene tetrachloride exposure. Removal of this man from exposure to this agent was followed by immediate recovery. A list of recommended precautions to be taken is included.

LAUGHLIN, ROBERT C. Continued exposure of human eyes to H vapour. M.I.T. subjects. (Chemical Warfare Service, Edgewood Arsenal. Medical Research Laboratory Report 9.) Off. Pub. Bd., Report, PB 9570. 1944. 9 pp. Price: Microfilm, 50c.; Photostat, $1.00.

The object of the investigation reported herein was to determine the type and extent of eye injury sustained by laboratory workers at M.I.T. who had developed eye symptoms from exposure to low concentrations of H vapour. Corneal changes, seen with the slit lamp, are found in a large percentage of subjects exposed to low concentrations of H vapour over a prolonged period. The initial impression is that these changes are mild and superficial, and will probably not lead to permanent injury.

LAUGHLIN, ROBERT C. Eye examination of factory workers handling H, CN and CG. (Chemical Warfare Service. Medical Research Laboratory. MRL(EA) Report 18.) Off. Pub. Bd., Report, PB 9576. 1944. 11 pp. Price: Microfilm, 50c.; Photostat, $1.00.

The eyes of all employees of the H, CN and CG plants were examined for conjunctival injection and corneal sensitivity, stained with fluorescein and examined with the slit lamp. Graphs and tables present data. Objective findings consisted of low-grade conjunctival injection, reduced corneal sensitivity, superficial punctate staining of the corneal epithelium, and pigmentation of the corneal epithelium. Follow-up examinations are recommended.

LAUGHLIN, ROBERT C. Miosis of the pupil as a test for water contamination by PF3. (Chemical Warfare Service. Medical Division Report 1.) Off. Pub. Bd., Report, PB 9505. 1944. 12 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Freshly prepared solutions of PF3 in water varying from 1 p.p.m. to 1,000 p.p.m. were instilled into the eyes of rabbits as single and as repeated instillations. A single instillation of 100 p.p.m. and repeated instillations of 20 p.p.m. or more caused definite miosis of the pupil within 15 minutes. Freshly prepared solutions of PF3 in water containing 25 p.p.m. and 50 p.p.m. were instilled into one eye of 20 men as single and repeated instillations. Three instillations of 25 p.p.m., or a single instillation of 50 p.p.m., caused moderate miosis within 30 minutes. Three instillations of 50 p.p.m. caused maximum miosis within 15 minutes. Miosis of the pupil is shown to be an adequately sensitive field test for PF3 in water.

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held on August 14, 1946, at the Children's Hospital, Melbourne, DR. A. P. DERHAM, the Chairman, in the chair.

Congenital Deformity of the Leg.

DR. A. MURRAY CLARKE presented a female child, aged four months, with a grossly deformed left leg, the result of congenital absence of the tibia. The knee was unstable, the fibula articulating with the side of the lower end of the femur. The foot was in a position of extreme inversion, the sole almost facing upwards. Dr. Clarke said that the patient was shown not so much because of any interest in diagnosis as to obtain an expression of opinion about the surgical management. There was a widespread belief that children could not or should not be fitted with artificial limbs, and consequently many varied and ingenious operations were done to save deformed and injured limbs. However, such authorities as Langdale-Kelham and George Perkins emphatically asserted that they knew from experience that children from the age of three and a half years could be fitted with artificial limbs, and were strongly of the opinion that they should. War injuries to children in England had provided many recent examples. Dr. Clarke said that just as a child learned to ski and to skate faster and better than an adult, so did he learn to walk with an artificial leg more quickly and more naturally. It was really remarkable how a child could move around on an artificial leg, and sometimes one could see a child running without being able to decide which leg had been lost. It was only necessary to see a child running with one leg and a crutch to realize how quickly the lost leg might "lose its place in the brain", and therefore how soon fitting of an artificial limb should follow amputation. Dr. Clarke said that he would therefore advocate amputation in this case when the child was aged between two and three years. Amputation could be preceded by the wearing of a Thomas walking calliper with a pelvic band and possibly shoulder straps, or a laced top if any ischial irritation occurred. The amputation should be a disarticulation through the knee joint in order to preserve the epiphysis at the growing end of the bone. The limb should be of the ischial weight-bearing type to prevent strain on the epiphysis. The question of expense was a very real one, and while a peg-leg would be light and cheap and satisfactory, limbs were now being made for children in a telescopic fashion so that they could be lengthened every six months and the rings could also be adjusted. This appreciably reduced the expense.

DR. JOHN JENS said that the case provided an interesting problem from the amputation point of view. The first point was that amputation was to be carried out on a small child. Experience during the "blitz" in England had shown that young children were good subjects for amputation, being very adaptable. The only drawbacks were the expense of maintenance of the artificial limb, which might be broken in games, and the expense of changing the limb every six months or so to allow for growth. Another point to be considered was the site of amputation. Until recently a through-the-knee operation was frowned on in British circles. War experiences had changed this view, and both British and American experiences proved that this type of amputation was good. A disadvantage reported by limb fitters was the broad, bulbous, distal end. This operation also necessitated an outside joint, which was not good for the lower limb. If amputation was carried out through the knee, the limb would lose a certain amount of length by pressure on the lower epiphysis. Lastly, Dr. Jens said that the tuber-bearing limb was something to which one could work back. The tuber was not always a permanency. The usefulness was impaired by scars, boils and other infections developing at the site.

Dr. Clarke, in reply, said that criticism of the tuber as an unsatisfactory site for weight-bearing had occurred to him, but he had omitted to mention it. A leather fitting jacket might avoid pressure on the tuber.

Cleido-Cranial Dysostosis.

DR. HENRY J. SINN said that the condition which he wished to bring to the attention of the meeting had been described under several names, the best known of which was hereditary cleido-cranial dysostosis. This name had been given to it by two Frenchmen, Marie and Sainton, in 1897. In an issue of *The Lancet* in 1910, Fitzwilliams had analysed the records of 58 cases and added two of his own. In 1929 Fitchett had surveyed the literature in *The Journal of Bone and Joint Surgery*. Dr. Sinn said that no doubt cases had been presented to the society, but not within his memory. This and the comparative rarity of the condition stimulated him to present this example. Dr. George Reid had described two interesting cases in young males in *The Royal Melbourne Hospital Clinical Reports* in 1940; one of these had been reported previously by Davis and Flecker in THE MEDICAL JOURNAL OF AUSTRALIA in 1928, when the patient was five years old.

Dr. Sinn then briefly outlined the main features of the syndrome. He said that the name "hereditary cleido-cranial dysostosis" suggested a bony dystrophy affecting the clavicles and the skull, and these indeed were the predominant findings, though the disease was by no means confined to these bones; nor was it necessarily hereditary. Aplasia of the clavicles was the commonest finding; it varied considerably in degree. Sometimes the whole and sometimes portion of the clavicles or of a clavicle—for the disease was curiously asymmetrical in some cases—was missing. This clavicular underdevelopment was responsible for the most striking clinical feature, the ability to approximate the shoulders. The same applied to the skull changes, which might be slight or pronounced. The usual findings were defective ossification of the calvarium as distinct from the base of the skull and face. The results were delayed closure of the sutures and unduly open fontanelles. Dentition might or might not be disturbed. The commonest finding was delay in eruption of the deciduous teeth and their late shedding. The permanent teeth were also late in appearing. The bones of the face might be underdeveloped and small; the palate might be narrow and highly arched. Associated bony abnormalities might be met with in the pelvic bones, spinal column, thorax and small joints of the fingers. There might or might not be deficiency of the muscles attached to the shoulder girdle. The ætiology was unknown; but it was thought to be an abnormality of the germ plasm itself. Diagnosis was not difficult once the syndrome had been seen. It had to be remembered that all degrees of the anomaly were met with. Confusion with hydrocephalus was the greatest danger, but the normal development ruled this out. The prognosis for life and normal mental development was good. No treatment was known, if indeed any was required, except to remember the increased vulnerability of the head to trauma because of the defective bone formation.

Dr. Sinn then showed a female child, aged twelve months, who had been referred to the out-patient department from the local baby health centre because the skull was not "knitting". She had been born one week prematurely and had weighed six pounds twelve ounces. Her mother had enjoyed good health during pregnancy, and delivery had been normal. The mother was struck by the unusual shape of the head soon after birth. The baby had a brother, aged three years, who was normally developed. Her mother and father were alive and well. There was no consanguinity in the family, and no abnormalities were present as far as could be learnt. The baby had been fed on the breast for three months and then on a cow's milk mixture, adequately supplemented with vitamins. She gained control of her head at three months, sat up at five months, crawled at eleven months, and was now at twelve months standing with support. Her hearing, sight and speech were normal for her age. On examination of the child, the head was seen to be large and square. It measured eighteen inches in circumference, and the suture lines were widely separated and the fontanelles unduly open. The chest was broad and deep, but the shoulders could be approximated with comparative ease. Soft rudimentary clavicles could be felt at the acromial ends, ending in rather a sharp point about three-eighths of an inch from the acromion. No teeth had as yet erupted. The results of the remainder of the examination were within normal limits. The diagnosis of cleido-cranial dysostosis was confirmed by radiological examination. X-ray films were taken of the head and chest. The extensive defects in bone formation of the cranial vault were laid bare, and it was impossible to recognize any bony deposits in the region of the clavicles. Defective bone formation at the pubes was also strikingly revealed.

DR. H. BOYD GRAHAM said that there was little to add to Dr. Sinn's description. The case was rare. Little was known of the anomaly beyond the information given in the case of the child presented. Dr. Graham said that he thought hypertelorism was present in this case.

DR. JOHN B. COLQUHOUN complimented Dr. Sinn on making the diagnosis. He said that as a rule such patients were brought under medical notice because of some other defect or disease. These children developed normally. They were sometimes the subject of exhibitions and gained notoriety

in that respect. One point had not been mentioned; this was a curious softening of the neck of the femur producing *coxa vara* and pronounced knock-knee. Anomalies were also seen at the epiphyses of the small bones of the hands. The condition was not as rare as was imagined. He had viewed an X-ray film of a boy similarly afflicted. Amongst other things, this film had shown curious changes in the skull called the Wormian phenomenon. The child's father displayed similar radiological findings.

DR. H. DOUGLAS STEPHENS congratulated Dr. Sinn on an unusual case. He said that he had very little to add. He had shown a patient to Dr. F. Wood Jones, who was then professor of anatomy at the University of Melbourne; this child "slipped through the hands"—this was one of the diagnostic features. Dr. Wood Jones had commented that cats did not possess clavicles.

(To be continued.)

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 5, of January 9, 1947.

PERMANENT NAVAL FORCES OF THE COMMONWEALTH (SEA-GOING FORCES).

To be Surgeon Captain.—Surgeon Commander (Acting Surgeon Captain) Lionel Lockwood, M.V.O., D.S.C.

AUSTRALIAN MILITARY FORCES.

Australian Army Medical Corps.

QX6439 Lieutenant-Colonel N. H. Morgan is transferred to the Reserve of Officers (Australian Army Medical Corps), 5th October, 1946.

VX114273 Lieutenant-Colonel (Temporary Colonel) J. J. Black, D.S.O., V.D., relinquishes the rank of Temporary Colonel, is placed upon the Retired List and is granted the honorary rank of Colonel with permission to wear the prescribed uniform, 22nd August, 1946.

N100925 Lieutenant-Colonel C. O. Donovan is placed upon the Retired List with permission to retain his rank and wear the prescribed uniform, 11th October, 1946.

VX66201 Major K. J. Eager is placed upon the Regimental Supernumerary List, 25th September, 1946.

NX191425 Captain D. D. Bathgate is seconded in the Active Citizen Military Forces for service with the Australian Imperial Force, 20th June, 1946.

QX6254 Major (Temporary Lieutenant-Colonel) R. R. Winton is placed upon the Regimental Supernumerary List, 1st November, 1946.

SX34115 Captain I. G. Pavy is removed from the Regimental Supernumerary List, 4th November, 1946.

Inter-Service Medical Wing Demobilization Centres (Australian Military Forces Component).—NX207659 Captain J. J. G. McGirr is removed from the Regimental Supernumerary List, 4th November, 1946.

To be Temporary Major, 28th June, 1946.—SX33991 Captain M. W. Elliott.

Reserve of Officers.

The undermentioned officers are transferred to the Reserve of Officers with effect from the dates indicated, and on the date prior to such transfer, where applicable, cease to be seconded. Officers holding temporary rank relinquish such temporary rank on the date of transfer to the Reserve of Officers.

Captains NX115821 T. W. Wiley, 24th September, 1946, WX39609 H. J. Hoffman, 3rd October, 1946, VX65536 J. M. McCracken, 5th October, 1946, and N429367 G. J. Miller, 26th September, 1946.

101st Australian General Hospital (Australian Imperial Force).—NX174912 Captain (Temporary Major) J. C. Dick, 5th October, 1946, and NX70989 Captain W. J. Skinner, 24th September, 1946.

No. 105 *(Adelaide) Military Hospital.*—Captains SX32353 W. Salter, 28th September, 1946, and SX33401 F. Bauer, 3rd October, 1946.

107th Australian General Hospital.—TX13148 Captain (Temporary Major) J. A. Oliphant, 5th September, 1946.

No. 112 *(Brisbane) Military Hospital.*—Captains QX57647 H. M. Whyte, 20th September, 1946, and NX206851 C. J. R. Conacher, 19th September, 1946.

No. 113 *(Concord) Military Hospital.*—Captains NX166708 H. E. Maderna, 21st September, 1946, NX200495 K. F. H. Hume and NX203566 J. R. Waddell, 24th September, 1946, and NX166625 T. Allen, 26th September, 1946.

No. 115 *(Heidelberg) Military Hospital.*—Captains VX90981 A. C. Reith, 2nd October, 1946, VX 90731 A. J. T. Aram, 3rd October, 1946, VX90978 I. H. Chenoweth, VX92708 J. P. Sullivan and VX94600 H. W. Ireland, 4th October, 1946, and VX91859 W. R. Spring, 5th October, 1946.

118th Australian General Hospital (Australian Imperial Force).—QX57863 Captain O. W. Salkeld, 27th September, 1946.

121st Australian General Hospital (Australian Imperial Force).—SX11585 Major R. A. Isenstein, 28th September, 1946.

14th Australian Camp Hospital.—NX131873 Captain J. M. Allingham, 19th September, 1946.

80th Australian Camp Hospital.—TX6275 Captain J. V. McGrath, 4th October, 1946.

111th Australian Convalescent Depot.—SX27916 Captain R. C. Heddle, 27th September, 1946.

Inter-Service Medical Wing Demobilization Centres (Australian Military Forces Component).—Captains NX77349 G. P. Dynon, 25th September, 1946, VX90033 F. E. Heymanson, 3rd October, 1946, and SX26061 R. V. Southcott, 3rd October, 1946, NX100089 Major A. K. Jones, 21st September, 1946, and QX33680 Captain J. W. Woodburn, 26th September, 1946.

NX34953 Captain J. W. McNamara, 10th July, 1946.

Captains TX6487 T. G. Ingram, 30th October, 1946, and NX203558 A. H. McGeoch, 23rd October, 1946.

2nd/11th Australian General Hospital.—NX147120 Captain W. L. H. Keller, 29th October, 1946.

No. 112 *(Brisbane) Military Hospital.*—VX64079 Captain J. R. Morris, 19th October, 1946.

No. 113 *(Concord) Military Hospital.*—NX203332 Captain (Temporary Major) D. M. Ross, 18th October, 1946, and NX201581 Captain I. G. Simpson, 25th October, 1946.

No. 115 *(Heidelberg) Military Hospital.*—Captains VX91030 J. J. Billings, 22nd October, 1946, and TX15383 H. C. Purton, 29th October, 1946.

130th Australian General Hospital.—VX66201 Major K. J. Eager, 23rd October, 1946.

15th Australian Camp Hospital.—NX76543 Major F. O. B. Wilkinson, 18th October, 1946.

52nd Australian Camp Hospital.—SX22157 Captain L. Bonnin, 31st October, 1946.

89th Australian Camp Hospital.—SX34384 Captain J. S. Anderson, 31st October, 1946.

2nd Australian Women's Hospital.—QX23691 Major R. A. Douglas, 24th October, 1946.

1st Australian Out-Patients' Depot.—NX200337 Captain W. N. C. G. Bennett, 25th October, 1946.

2nd/1st Australian Hospital Ship.—QX46550 Captain L. N. Ferrari, 18th October, 1946.

Inter-Service Medical Wing Demobilization Centres (Australian Military Forces Component).—Captains NX204410 F. G. J. Clarke, 19th October, 1946, VX114281 M. H. Smith, 15th October, 1946, and WX41116 J. R. Ambrose, 1st November, 1946.

QX5236 Lieutenant I. R. Cameron, 19th October, 1946.

SX19025 Captain J. M. McPhie, 17th October, 1946.

2nd/11th Australian General Hospital.—Captain NX146280 J. F. McInerney, 1st October, 1946, and NX111972 C. Y. Symons, 1st October, 1946.

2nd/12th Australian General Hospital.—NX277402 Major J. F. Lipscombe, 14th September, 1946.

101st Australian General Hospital (Australian Imperial Force).—NX77358 Captain L. V. Church, 27th September, 1946.

No. 113 *(Concord) Military Hospital.*—NX76435 Major E. Murray-Will, 4th October, 1946, Captains NX207587 R. M. Gibson, 27th September, 1946, NX129617 J. P. Ryan, 2nd October, 1946, NX202351 J. H. Findlater, 4th October, 1946, and NX204416 G. C. Wilson, 15th October, 1946.

No. 115 *(Heidelberg) Military Hospital.*—VX91438 Captain H. McL. Stevenson, 10th October, 1946.

120th Australian Special Hospital.—NX76364 Captain C. E. Marshall, 1st October, 1946.

16th Australian Camp Hospital.—NX99731 Captain K. R. Barder, 10th October, 1946.

70th Australian Camp Hospital.—NX114070 Captain B. H. Dolman, 1st October, 1946.

Inter-Service Medical Wing Demobilization Centres (Australian Military Forces Component).—Captains NX201215 R. B. Wiles, 27th September, 1946, NX200475 V. H. Hegarty, 10th October, 1946, and NX149382 A. T. Clements and NX202318 L. A. Wherrett, 1st October, 1946.

NX444 Captain E. V. Barling, 28th May, 1946.

The undermentioned officers are transferred to the Reserve of Officers on the dates indicated, and, where applicable, they cease to be seconded. Officers holding temporary rank relinquish such temporary rank on the date of transfer to the Reserve of Officers and are granted from such date honorary rank on the Reserve of Officers equivalent to the temporary rank relinquished.

No. 105 *(Adelaide) Military Hospital.*—SX33300 Captain (Temporary Major) D. G. McKay, 3rd October, 1946.

118*th Australian General Hospital (Australian Imperial Force).*—SX26261 Captain (Temporary Major) R. E. Britten-Jones, 26th September, 1946.

118th *Australian General Hospital (Australian Imperial Force).*—VX148899 Captain (Temporary Major) F. Catarinich, 12th October, 1946.

Inter-Service Medical Wing Demobilization Centres (Australian Military Forces Component).—NX77382 Captain (Temporary Major) R. B. Blacket, 27th September, 1946.

Retired List.

The undermentioned officers are placed upon the Retired List on the dates indicated with permission to retain their present substantive rank and wear the prescribed uniform. Where applicable they cease to be seconded, and relinquish any temporary rank held with effect from the date of placement upon the Retired List.

2nd Australian Blood and Serum Preparation Unit.—N271594 Major R. J. Walsh, 21st September, 1946.

TX6071 Captain G. M. Crabbe, 27th September, 1946.

WX11015 Major A. W. Farmer, 23rd October, 1946, and NX35139 Captain R. M. Mills, 24th October, 1946.

No. 113 *(Concord) Military Hospital.*—NX200830 Captain F. C. Hughes, 5th October, 1946.

121*st Australian General Hospital (Australian Imperial Force).*—SX33404 Captain D. L. Wilhelm, 12th October, 1946.

Reserve Citizen Military Forces.

2nd Military District.—Captain M. M. Dixon is placed upon the Retired List with permission to retain her rank and wear the prescribed uniform, 9th October, 1946.

3rd Military District.—Lieutenant E. U. Minchin (*née* Henniker) is placed upon the Retired List with permission to retain her rank and wear the prescribed uniform, 9th October, 1946.

5th Military District: To be Honorary Captain, 27th September, 1946.—William James Grey.

1st Military District.—The notification respecting Honorary Major F. G. Griffiths which appeared in Executive Minute No. 196 of 1946, promulgated in *Commonwealth Gazette,* No. 200, of 1946, is withdrawn.

2nd Military District.—The undermentioned officers are placed upon the Retired List with permission to retain their ranks and wear the prescribed uniform, 16th October, 1946: Captains B. J. McNamara and J. V. R. Chapman, Honorary Captain L. J. Colwell, Lieutenants L. R. Cohen and M. F. Filby.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force: Medical Branch.

The appointments of the following officers are terminated on demobilization with effect from the dates indicated: (Temporary Wing Commanders) C. J. Cummins (261205), F. S. Parle (261215), 19th November, 1946, (Flight Lieutenants) H. S. Moore (266931), 21st November, 1946, C. M. Maxwell (266828), 25th November, 1946, R. A. Russell (287441), 28th November, 1946.

Reserve: Medical Branch.

The following officers are appointed to commissions with the ranks as shown with effect from the dates indicated: (Temporary Wing Commander) Cyril Joseph Cummins (261205), Francis Sherlock Parle (261215), 20th November, 1946, (Temporary Squadron Leader) John Anthony Bond (261488), Charles Burgoyne Hudson (263255), Otto Henry Schneider (263716), Keith Franklin Drysdale Sweetman (252344), 1st December, 1946, (Flight Lieutenant) Ernest William Lee (266050), 1st December, 1946.—(Ex. Min. No. 292—Approved 8th January, 1947.)

Special Correspondence.

NEW ZEALAND LETTER.

FROM OUR SPECIAL CORRESPONDENT.

Visit of Sir William Fletcher Shaw.

SIR WILLIAM FLETCHER SHAW, official visitor to New Zealand from the Royal College of Obstetricians and Gynæcologists, has just left for Australia after three months in the country. The prime object of his visit was to advise the Auckland University College Council and the Auckland Hospital Board on matters pertaining to the newly endowed post-graduate chair of obstetrics and gynæcology, which has been established in Auckland. A fund, which has almost reached the objective of £100,000, has been raised throughout the country by a committee of Auckland business men over the last two or three years, and this is to be placed at the disposal of the Auckland University College Council for the appointment and management of the chair. A special hospital for obstetric and gynæcological cases has been promised by the Government, and it is hoped that within a short time some decision as to the exact location of the hospital will be made and the time of the beginning of its construction determined.

A series of conferences with the Board and the University Council has been held with Sir William Fletcher Shaw, and satisfactory progress has been made towards the objects in view.

In addition to attending to this main matter, Sir William Fletcher Shaw has travelled through the country, and his help on many contingent matters has been valuable. For example, before leaving Auckland he made statements for the Press on various hospital matters, particularly concerning hospital appointments, and also those in his own field of obstetrics and gynæcology. These have had a great effect in the right direction, and his help has been greatly appreciated.

CANADA LETTER.

FROM OUR SPECIAL CORRESPONDENT.

WITH the new year, Canada's first compulsory hospitalization plan has come into effect in the Province of Saskatchewan, where an estimated number of 800,000 individuals will be covered at $5.00 per person or $30.00 per family. Contracts are being let in that Province for a 600-bed teaching and diagnostic hospital on the campus at Saskatoon, and a neighbouring 1,200-bed mental hospital.

The number of doctors is increasing in Canada at a rate which, in 1951, will give the country one doctor per 852 people. The ratio in some cities is likely to be one per 500 residents, and if present trends continue, some rural areas will have one doctor per 10,000 scattered residents. During the war encouragement was given to doctors to go to the far north on a guarantee of the rank pay of a major in the medical corps, *plus* expenses. What incentive will be sufficient in peace time is hard to determine, but the need is great. Many denominational missions carry medical and nursing care to the isolated northern posts, and through mission boats to the settlers along the islands and inlets of the coasts.

Certification of specialists has been delegated to the Royal College of Physicians and Surgeons at Ottawa, who will hold their first diploma examinations in specialties in October, 1947. These will include: in surgery—neurosurgery, obstetrics and/or gynæcology, orthopædic surgery and urology; in medicine—dermatology and syphilology, neurology and psychiatry, and pædiatrics. Under any health insurance scheme in the future, and for workmen's compensation board work, it will soon be essential to have such a qualification for recovery of specialist fees.

A medical branch of the National Research Council at Ottawa has been set up under the chairmanship of Dr. J. B. Collip, Director of the Institute of Endocrinology of McGill University. An adequate budget is envisaged, and grants from this source may prove to be a determining factor in the calibre of medical teachers who can be retained in Canada in spite of the competition offered by better-paid positions in American institutions.

Medical standards for civil aviation are under consideration in this country, particularly because of PICAO's current meeting in Montreal. The recent epidemic of crashes throughout the world has brought the question of pilot error to the fore. Especially should there be searching of heart in the United States of America, where, despite opposition from the Aero-Medical Association, legislation was brought in whereby any general practitioner could certify a person fit to fly. It has been widely felt that this has been a disservice to aviation, and recent events have tended to confirm this.

To illustrate what well-selected and well-trained pilots can do, one might mention the record of the Air Ambulance Service in Saskatchewan. Recently one of its aeroplanes made eighteen mercy flights in six flying days, operating at times into unmapped and virgin territory. Another nurse has been added to the staff to help with the winter emergencies, which will increase rapidly now that snow storms are likely to block all ground communications when they are needed. The use of ski landing apparatus has brought the air ambulances right to the farmhouse door.

The recent visit to Montreal of the brilliant English biophysicist, W. Grey Walter, has brought forth a renewed interest in electroencephalography through the objective analysis of recorded "brain waves" on an automatic frequency analyser which he designed at the Burden Neurological Institute in Bristol. His work may revolutionize our ways of interpreting the wave forms seen on the miles of electroencephalographic records which daily are poured out in America. Now that the legal profession is taking up the electroencephalograph as a serious adjunct to medical evidence in court cases, there is an ever greater need for accuracy in interpretation.

Correspondence.

THE TREATMENT OF THYREOTOXICOSIS BY CONCURRENT ADMINISTRATION OF THIOURACIL AND IODINE.

SIR: In the letter by Sir Alan Newton in the issue of the journal of January 11, 1947, he states that he "felt that skilled physicians assisted by biochemists and other laboratory workers were better qualified to assess the value of the drug (thiourea)" than he was, and "being less bold" than I he handed a supply of this drug to the Royal Melbourne Hospital.

As Sir Alan was at that time very busily engaged on important national work in the war effort of our country, one could not expect him to concern himself actively in this new form of treatment in a field of surgery wherein he is a recognized master.

As regards the dangers associated with the use of thiourea and thiouracil, I thought in the various lectures and published articles for which I have been responsible and in the original article (THE MEDICAL JOURNAL OF AUSTRALIA, April 13, 1946) with S. L. Spencer, the risks were fully detailed and warning given as to the necessity for laboratory controls, and these warnings have been reiterated in the article he criticizes. I have fortunately been associated with expert biochemists and had full laboratory facilities with all my cases, but apparently, because I am a surgeon, I am not regarded as being so fully qualied as a physician to evaluate a form of medical treatment for a condition which I have studied for forty years. Was it not Lord Moynihan who referred to the operating physician as being the best surgeon? Yet Sir Alan Newton chides me for "becoming a little medical minded". Has Sir Alan Newton forgotten the early days of the sulphonamides and the gradual introduction of newer compounds, until today we have attained relative safety in the use of this group of drugs—surgeons as well as physicians? The same thing applies to the story of the acridines in the control and prevention of wound infections, so that I am not ashamed of "the enthusiasm with which Mr. Poate leaves that labelled thiouracil for that labelled methyl-thiouracil", for it is just as much an advance in therapy as was the step up from sulphanilamide to sulphadiazine, and already we have improved forms of "thio" drugs, such as propyl-thiouracil, which may put methyl-thiouracil in the background.

I regret that Sir Alan Newton was not clear on my statement that "it seems that permanent control of the thyreotoxic factor can be achieved in 85% of cases", as I state in conclusion that it is in primary hyperplastic conditions that these results are attained; also in the article on methyl-thiouracil I remark: "Satisfactory results can be obtained only in cases of acute hyperplastic toxic goitre." I was surprised to hear that Sir Alan Newton saw only 24% of such cases in his practice, as my own figure is very much higher, being close to 40%. I do not advise medical treatment of thyreotoxicosis associated with adenomata, either single or multiple, nor with toxicity supervening on an old colloid goitre—all such cases are referred for operation and in many of them use is made of thiouracil or methyl-thiouracil as a pre-operative measure, giving iodine for two to three weeks prior to operation. As to the mixed vitamin I advise—this is not "rule of thumb", as I use a standardized preparation in doses adjusted to the clinical needs of the patient and often supplemented by other vitamins of the *B* group.

From my experience in the treatment of thyreotoxicosis, I consider the surgeon and not the physician should have the full control of these cases, as even with the recently instituted medical treatment with "thio" compounds the surgeon is the one who can better determine whether or not surgery should be undertaken.

Yours, etc.,

225, Macquarie Street, Sydney,
January 15, 1947.

HUGH R. G. POATE.

THE ZOOLOGICAL POSITION OF MAN.

SIR: In your journal of December 14, 1946, a letter appeared criticizing my presidential address entitled "The Zoological Position of Man", which you had published on November 2, 1946. Owing to preoccupation with examinations, I had delayed replying to this anonymous letter, signed "Criticus". Meanwhile much of what I had intended to say in reply to "Criticus" has been said more ably and quite independently by my colleague, Professor A. A. Abbie, of Adelaide. However, there are still certain points of "Criticus's" letter to which I should like to reply briefly.

Firstly, as regards my appraisal of Huxley's book, "Evolution, the Modern Synthesis", this is a matter of opinion, and I am quite prepared to leave the decision to the future. Certainly, a perusal of the chapter headings and bibliography alone, will convince most observers that there is little lack of breadth, and that there are few men more capable of covering such an immense field with such perspicacity and accuracy.

Secondly, as regards the gene theory, I decided to take a definite standpoint, and accepted the theory as developed by Morgan, Huxley, Haldane, Fisher, Darlington *et cetera*, and deliberately rejected Goldschmidt's criticisms. Whether I was right or wrong, time alone will decide.

Thirdly, in discussing Huxley's thesis that "a brain capable of conceptual thought could not have been developed elsewhere than in a human body", while this may be open to argument, nevertheless, as Sherrington says, "the occurrence of mind—recognizable finite mind—is confined to a certain particular field of chemistry and physics, namely, that of highly integrated animal lives". In the same paragraph "Criticus" says: "Moreover, the majority of philosophers deny that the brain is capable of conceptual thought." This is surely an overstatement. While admittedly the problem of the relationship of body and mind remains unsolved, and very many philosophical theories have been put forward concerning this relationship, nevertheless what Keats dubbed "busy common sense" certainly gives us the right to correlate mental experience and conceptual thought with the activities of the human roof brain or cerebral cortex (contrast Sherrington, "The Brain and its Mechanism"). Elsewhere, referring to this relationship between body and mind, Sherrington remarks: "But today, less than yesterday, do we think the definite limits of exploration yet attained"; and yet again he says: "I am not a defeatist, for I would urge active pursuit of the inquiry." It is, then, I think, a fair working hypothesis, nowadays, that conceptual thought or mental experience on the one hand, and human brain happenings on the other, coincide in time and space, to paraphrase Sherrington again. In conclusion, it seems clear that while several of the points raised by "Criticus" result from misunderstanding or misreading of my address, such as numbers 13, 15 and 16, as Professor Abbie has so clearly pointed out, others are due to a different selection of and emphasis upon present-day theories, and here I prefer my own judgement to that of "Criticus".

Yours, etc.,

Department of Anatomy,
University of Sydney,
January 20, 1947.

A. N. BURKITT.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales, as duly qualified medical practitioners:

Newman, Neville Maurice, M.B., B.S., 1946 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
O'Dea, Bettine Frances, M.B., B.S., 1946 (Univ. Sydney), St. George District Hospital, Kogarah.
O'Flynn, Patricia Mary, M.B., B.S., 1946 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
Ormiston, Roslyn Isabel, M.B., B.S., 1946 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Percy, Neville, M.B., B.S., 1946 (Univ. Sydney), Sydney Hospital, Sydney.
Phillips, John Bertram, M.B., B.S., 1946 (Univ. Sydney), Sydney Hospital, Sydney.
Prior, John, M.B., B.S., 1946 (Univ. Sydney), Lewisham Hospital, Lewisham.
Richardson, Stephen, M.B., B.S., 1946 (Univ. Sydney), Balmain and District Hospital, Sydney.
Robilliard, Joseph Arthur, M.B., B.S., 1946 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Rogers, Peter Augustine, M.B., B.S., 1946 (Univ. Sydney), Mater Misericordiæ Hospital, North Sydney.
Rossell, Margaret Sinclair, M.B., B.S., 1946 (Univ. Sydney), the Prince Henry Hospital, Little Bay.

Obituary.

DALLAS BRADLAUGH WALKER.

WE regret to announce the death of Dr. Dallas Bradlaugh Walker, which occurred on January 10, 1947, at Rockhampton, Queensland.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Manzie, Peter Podmore, M.B., B.S., 1945 (Univ. Sydney), Newcastle General Hospital, Newcastle.

The undermentioned, registered by the Medical Board of Victoria under the provisions of the *Medical Practitioner's Registration Act*, 1946, have applied for election as members of the Victorian Branch of the British Medical Association:

Bettinger, Hans Frederick, M.D. (Univ. Breslau), Department of Pathology, the Women's Hospital, Carlton.
Brunton, Mark George, M.D. (Czecho-Slovakia), 20, The Esplanade, St. Kilda.
Huppert, Isodore, M.D. (Univ. Prague), 6, William Street, North Brighton.
Jakobowicz, Rachel, M.D. (Univ. Berlin), Flat 7, 129, Brighton Road, Elwood.
Karoly, Margaret, M.D. (Univ. Budapest), 14, Ross Street, Toorak.
Landauer, Friedrich Max, M.D. (Univ. Strassburg), Murrayville.
Schuller, Arthur, M.D. (Univ. Vienna), 33, Collins Street, Melbourne.
Singer, Kora Renata, M.D. (Univ. Vienna), 10, Bradford Avenue, Kew.
Suss, Max Manfred, M.D. (Univ. Vienna), Tongala.

Books Received.

"The Centennial of Surgical Anesthesia. An Annotated Catalogue of Books and Pamphlets Bearing on the Early History of Surgical Anesthesia Exhibited at the Yale Medical Library, October, 1946", compiled by John F. Fulton, M.D., and Madeline E. Stanton, A.B.; 1946. New York: Henry Schuman. 9¼" × 6¼", pp. 118, with illustrations. Price: $4.00.

"A Memoir to the Academy of Sciences at Paris on a New Use of Sulphuric Ether", by W. T. G. Morton, of Boston in the U.S.A., Presented by M. Arago in the Autumn of 1847, with a Foreword by John F. Fulton; 1946. New York: Henry Schuman. 9¼" × 6¼", pp. 30. Price: $1.50.

"Principles in Roentgen Study of the Chest", by William Snow, M.D.; 1946. Springfield, Illinois: Charles C. Thomas. 10" × 6½", pp. 424, with many illustrations. Price: $10.00.

Diary for the Month.

FEB. 4.—New South Wales Branch, B.M.A.: Organization and Science Committee. Special Groups Committee.
FEB. 5.—Victorian Branch, B.M.A.: Branch Meeting.
FEB. 5.—Western Australian Branch, B.M.A.: Council Meeting.
FEB. 7.—Queensland Branch, B.M.A.: Branch Meeting.
FEB. 8.—Tasmanian Branch, B.M.A.: Annual Meeting.
FEB. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
FEB. 14.—Queensland Branch, B.M.A.: Council Meeting.
FEB. 17.—Victorian Branch, B.M.A.: Finance Meeting.
FEB. 18.—New South Wales Branch, B.M.A.: Medical Politics Committee.
FEB. 20.—Victorian Branch, B.M.A.: Executive Meeting.
FEB. 25.—New South Wales Branch, B.M.A.: Ethics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute; Brisbane City Council (Medical Officer of Health). Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

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